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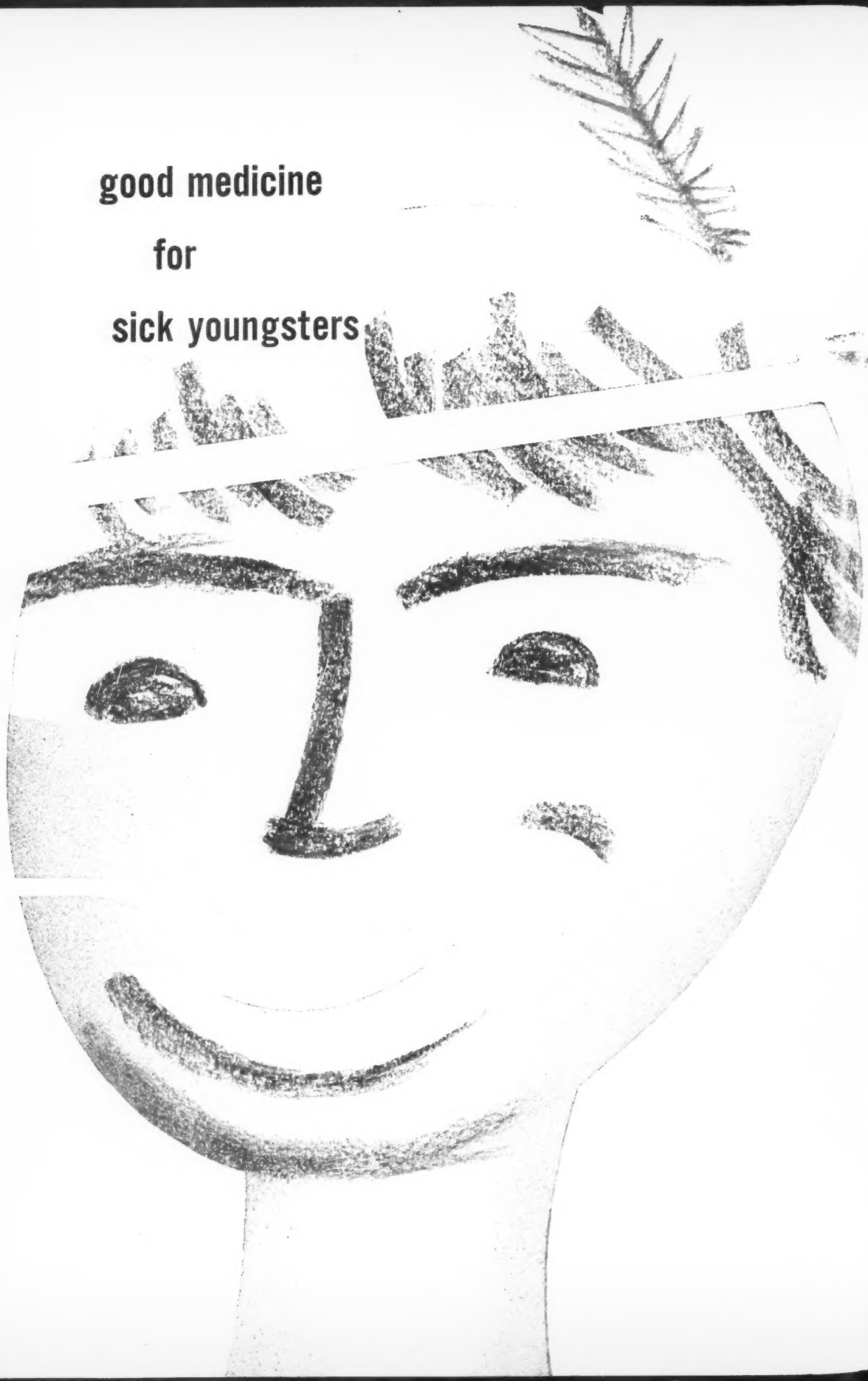

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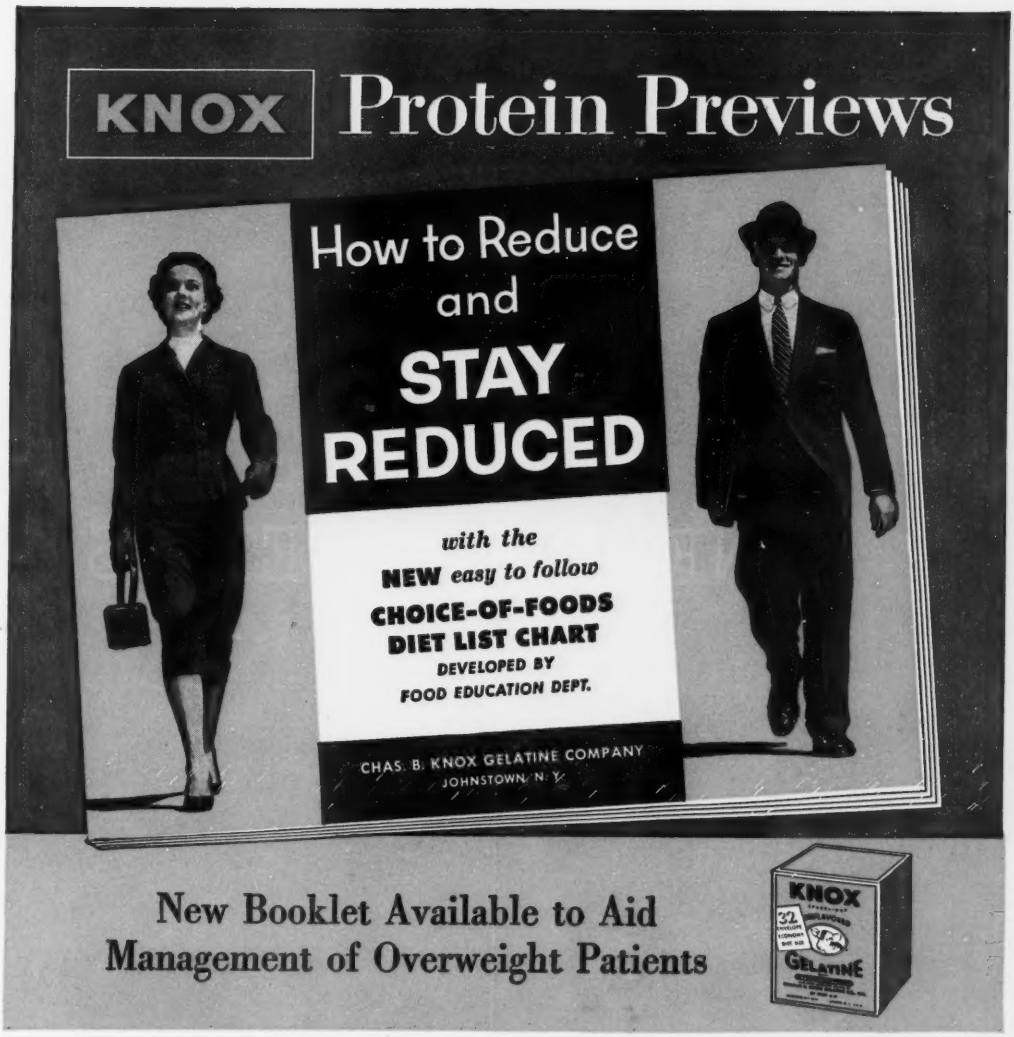

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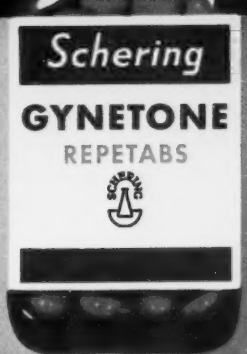

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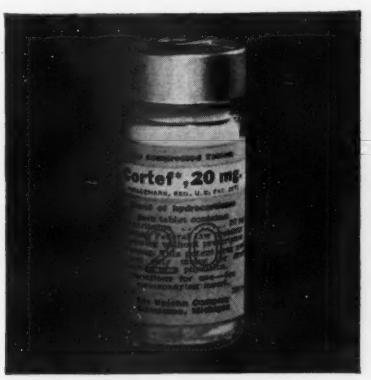

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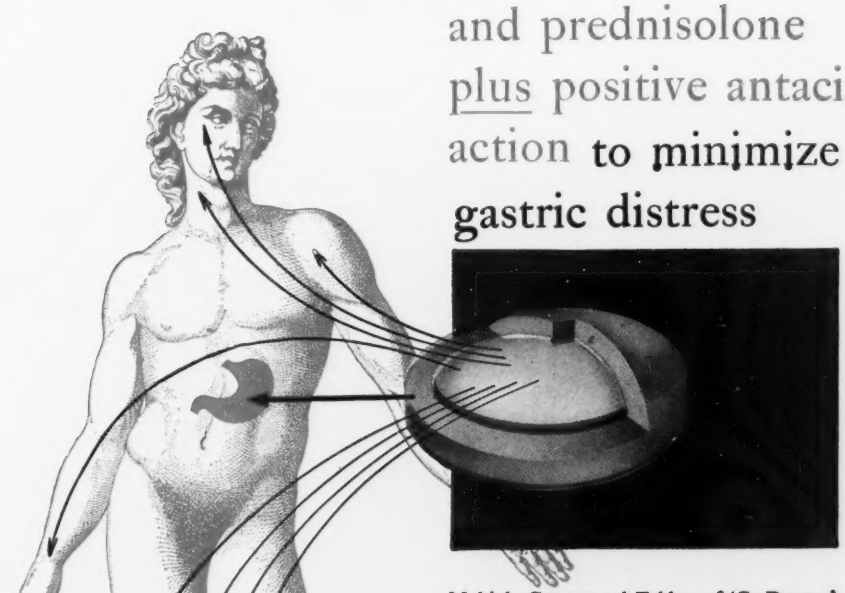

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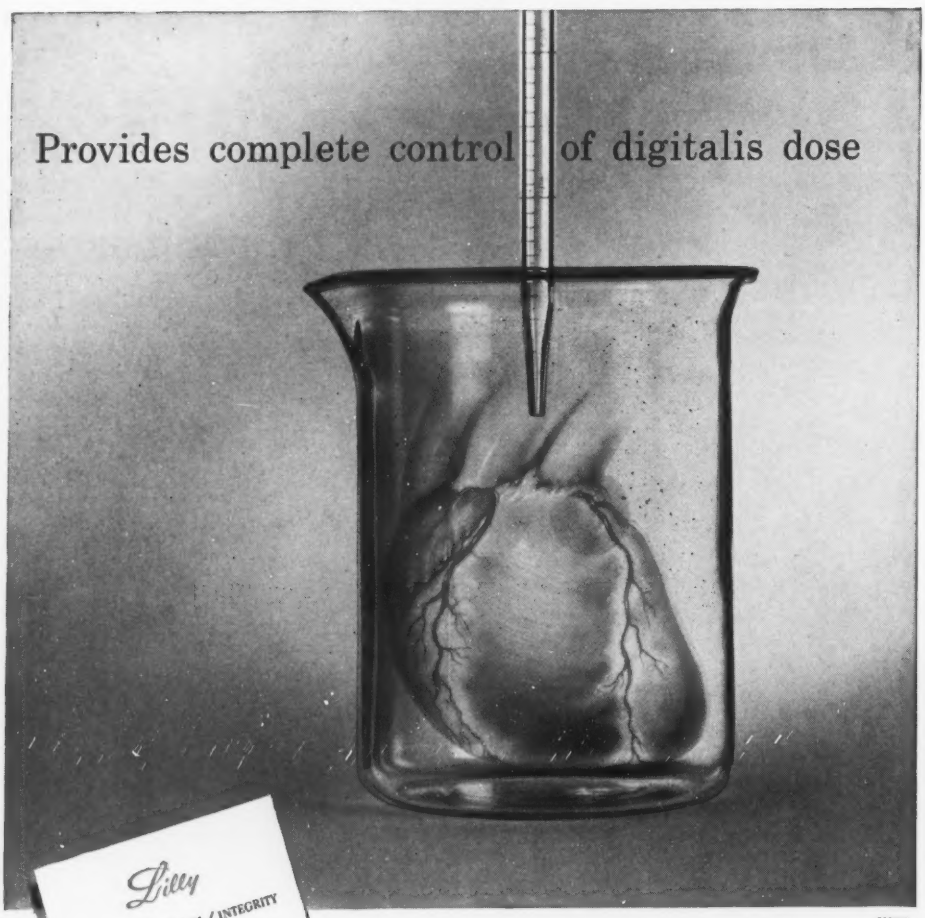

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Volume LVII **MAY, 1956** **No. 5**

Pregnancy and Heart Disease

The Surgical Correction of Mitral Stenosis in the Presence of Pregnancy

JOHN G. SHELLITO, M.D., *and* JOHN K. FULTON, M.D., *Wichita*

Editor's Note. This article and the one following were, through coincidence, received by the Journal at almost the same time and are being published together even though they do not necessarily reflect identical ideas. The problem of the pregnant woman with a complicating mitral stenosis is discussed, with consideration of a possible surgical attack on the heart lesion rather than therapeutic abortion or continuation of a complicated pregnancy.

We are indebted to Bailey, Glover, and O'Neil,[1, 2] together with Harkin,[10] for our knowledge of early surgery of the mitral valve.

Glover[7] has pointed out that organic heart disease, usually rheumatic, complicates 1 to 2 per cent of all pregnancies.

Categorization of patients referable to the severity of their disease has now been established. Group I comprises asymptomatic patients. These are persons with ausculatory findings of mitral stenosis who have no symptoms referable to their disease. There is no enlargement of the heart and there is no specific electrocardiographic change.

Group II consists of patients who have symptoms referable to the heart but have never had congestive failure.

Group III consists of patients who have had congestive failure, who show the signs and symptoms of the Group II patient, but who also have systemic congestive manifestations which can be controlled.

Group IV includes patients who have intractable congestive heart failure due to mitral stenosis.

It is now known that surgery is much better tolerated by patients in Groups II and III than in Group IV. Surgery is not usually necessary in Group I.

To date the number of reported mitral commissurotomies during pregnancy has been small. Cooley and Chapman[5] reported two cases successfully operated in the fourth month and 36th week of pregnancy, respectively. Baker et al.[3] reported three cases, one performed in the third month and two in the 28th week. Watt, Bigelow, and Greenwood[13] reported seven cases operated on during the first or second trimester of pregnancy. Three had been delivered at the time of the report, and all had shown "continued improvement" in cardiac status following the operation.

Glover et al.[7] published a report of their experience with five mitral commissurotomies during pregnancy. Four of the patients delivered normal infants following commissurotomy. One of the five patients had a spontaneous abortion six weeks following surgery. All were improved in the postpartum period.

The decision to undertake mitral commissurotomy during pregnancy is not to be lightly considered. In cases thus far reported the indications have not always been clearly outlined. The preoperative condi-

From the Departments of Surgery and Internal Medicine, Wichita Clinic, Wichita, Kansas.

tions of those described by Watt et al.[13] are not given in detail. One had had repeated episodes of failure and hemoptysis before her pregnancy. The patient reported by Cooley and Chapman[5] had been in congestive failure shortly before the procedure. Rhythm was regular and the response to a preparatory medical regimen had been good. Each of the three patients reported by Baker et al.[3] had been subject to attacks of pulmonary edema either before or after onset of pregnancy. These authors advocate mitral commissurotomy as an emergency measure if intractable failure occurs in the second or third trimester of pregnancy.

Alternatively, medical management must be evaluated as to its efficacy and safety in dealing with the situation. Mortality from medical management of Grade II cardiacs followed by MacRae[11] was 1.4 per cent, utilizing bed rest in management and digitalis, salt restriction, and diuretics when necessary. Barry[4] reported a maternal mortality of 0.37 per cent in his Grade II cardiacs and a figure of 1.42 per cent in Groups III and IV. Hamilton[9] reports a mortality figure of 3 per cent, and Stromme and Kuder[12] report a figure of 1.3 per cent. Such statistics speak well for medical management in most instances where pregnancy is complicated by cardiac disease.

Mortality in large series of mitral commissurotomies is usually stated to be 6 per cent. On statistical grounds, mitral commissurotomy is not the treatment of choice for the usual case of mitral stenosis associated with pregnancy. It is also doubtful, in view of the above figures, that therapeutic abortion is often indicated on the basis of anything but the most severe and intractable cardiac disease. It should be pointed out that older series on mortality of the cardiac patient in pregnancy gave maternal mortality figures as high as 18 per cent for Grade III patients. With modern meticulous attention to sodium restriction, rest, diuretics, and digitalis, the figure has been much improved, as the several mentioned series indicate.

The decision to perform mitral commissurotomy on our patient was reached on the basis of several considerations: (1) She was considered an ideal candidate for the procedure, had pregnancy not been present; (2) Her previous pregnancy had been marked by complete disability with repeated hemoptyses; (3) When non-pregnant, her compensation was excellent without restriction of activities, and (4) Economically she was unable to provide for herself the required rest and other attentions necessary for adequate medical management.

CASE REPORT

Mrs. G. L., C-107,401, 24-year-old white gravida III, para IV, was first seen in the first month of pregnancy. During her last pregnancy 18 months previously she had had ankle edema, ease of fatigue, occasional hemoptyses, and marked dyspnea to such a degree that she was bedfast during the entire pregnancy. Two weeks postpartum she had regained her usual exercise tolerance and was able to climb stairs without dyspnea. She first suspected pregnancy when she noted the return of dyspnea and fatigue. Physical findings showed a normal rhythm, slight cardiac enlargement, a prominent systolic and presystolic thrill at the apex, and an easily palpable thrust synchronous with P2 at the base. Typical rumbling diastolic and soft systolic murmurs, together with a loud snapping first apical and loud second pulmonic sound, were heard. The blood pressure was 100/60 mm. There was no edema or distention of the neck veins.

On December 1, 1954, utilizing the usual anterolateral incision through the bed of the resected fourth rib, the chest was opened. When the auricular appendage was touched there were immediate aberrant contractions of the cardiac musculature. Procaine was utilized for the insertion of the purse string suture, and some intravenous procaine was given by the anesthetist. Entrance to the left auricle was via the appendage in the classic fashion. A typical mitral stenosis was found, having an opening approximately the size of the head of a kitchen match. The anterolateral commissure was thoroughly split and dilated to the edge of the valve ring. The posterolateral commissure was not split or cut.

Pathological examination of the resected portion of the auricular appendage demonstrated a small Aschoff nodule.

The patient stood the procedure well and was dismissed from the hospital nine days postoperatively. She has since been delivered of a normal infant without difficulty or evidence of cardiac failure.

Most certainly the case reported is not representative of what is to be expected of mitral commissurotomy as a procedure of choice for mitral stenosis encountered during pregnancy. Some will have large hearts with weakened myocardiums, some will have irreversible pulmonary changes, some will have auricular fibrillation, calcified valves, or previous history of embolization suggesting that the mortality from mitral commissurotomy would exceed the expected benefits. Many others, while having undoubted mitral stenosis, will tolerate pregnancy without marked difficulty, as was the case with this patient in her first two pregnancies.

COMMENT

1. Uncomplicated pregnancy associated with mitral stenosis is not a contraindication to commissurotomy.

2. The commissurotomy should be done during the first trimester of pregnancy, if possible.

3. Mitral commissurotomy during pregnancy should be reserved for those patients who otherwise would require therapeutic abortion. These are patients who can be expected to develop intractable failure or hemoptysis or cannot cooperate with a medical regimen.

4. Cardiacs who can be expected to remain in category I or II during pregnancy may delay commissurotomy until after delivery, if it is then indicated.

The Wichita Clinic
3244 East Douglas
Wichita, Kansas

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Pregnancy and Heart Disease

Mitral Commissurotomy during Pregnancy

ALFRED M. TOCKER, M.D., ERNEST W. CROW, M.D., *and* GRANT E. EVANS, M.D., *Wichita*

Although mitral commissurotomy is now widely accepted and firmly established as a standard surgical intervention with low mortality and good results, the utilization of this procedure in the pregnant woman presents special problems. There are now numerous short reports of mitral commissurotomy during pregnancy[9, 25] which have shown that the hazards of mitral surgery are more apparent than real. These have proved that even in severe cases of mitral stenosis, pregnancy need not invariably be terminated. Since the long term results of the operative procedure itself, as well as its relation to pregnancy, will not be known for perhaps another decade, policies with regard to utilization of this surgical procedure under such circumstances must be tentative.

Asymptomatic lesions, often unknown to the patients, are discovered during routine prenatal physical examinations. In about half the cases so discovered in one clinic, the patients were not aware of the existence of the disease.[7] The effectiveness of case finding in this group of patients is further enhanced by the effect of pregnancy in making the signs of heart disease more obvious. Obstetrical patients with borderline indications for surgery, if they can be carried through their pregnancies safely, will often show a minimum of signs and symptoms in the non-pregnant state.

Yet the fact must be kept in mind that in the practice of obstetrics, heart disease is one of the leading causes of maternal morbidity and mortality. Organic heart disease complicates 1 to 2 per cent of all pregnancies[11] and accounts for about 25 per cent of all maternal deaths.[6] Ninety per cent or more of the cardiac lesions that occur in pregnant women are attributable to rheumatic etiology,[21] and in at least two-thirds of these patients there is involvement of the mitral valve alone, usually with mitral stenosis with or without mitral insufficiency.[7, 11, 15] Eighty-five per cent of these rheumatic heart disease patients are in functional Class I or Class II[23] (New York Heart Association Classification) and generally tolerate their pregnancies well.

From the Departments of Surgery, Internal Medicine, and Obstetrics and Gynecology, Wesley Hospital, Wichita, Kansas.

In unfavorable cases, maternal mortality approaches 40 to 45 per cent, and cardiac decompensation accounts for 60 to 65 per cent of the fatalities.[21] This relatively small group of patients with severe mitral stenosis (Classes III and IV) accounts for most maternal mortality, and it is in this group that one must choose between continuation of the pregnancy, interruption by curettage or hysterotomy, or mitral commissurotomy.

Some time ago we were faced with such a decision. It is the purpose of this paper to present this case history and discuss the question, pro and con, of mitral commissurotomy during pregnancy.

CASE HISTORY

Mrs. G. T., a 27-year-old white housewife, was admitted to Wesley Hospital, Wichita, on August 17, 1953, with a diagnosis of threatened abortion and inactive rheumatic heart disease, predominantly mitral valvulitis with stenosis. She was estimated to be approximately in her third month of pregnancy.

Past history revealed that at the age of 12 she had had uncontrollable muscle jerkings and was treated as a bed patient for six months. Since childhood she had been subject to aching of her legs with severe cramps and coldness of her feet and frequent upper respiratory infections. Her best weight had been 106 pounds some six years previously, and she had lost weight, weighing 84½ pounds on admission. Three times since age 19 she had experienced fainting after undue exertion and had had progressive exertional dyspnea for six years to the point of dyspnea on walking less than one flight of stairs. Slight dependent edema had been present for four years intermittently. Auricular fibrillation appeared in May 1953. She was digitalized April 1953, at which time commissurotomy was first advocated. She stated she always felt tired, whether or not she exerted herself. Although she felt she was able to do her own housework prior to her pregnancy if she took her time, for some time her mother had been doing her housework for her.

The patient was a para I, gravida V. Her first pregnancy had resulted in the birth of a premature infant, who died shortly after birth. Her second and third pregnancies resulted in miscarriages. Her last pregnancy six years ago terminated favorably with the delivery of a male infant weighing 5 pounds and 5 ounces. Although she weighed 104 pounds at the onset of her fourth pregnancy, she weighed only 96

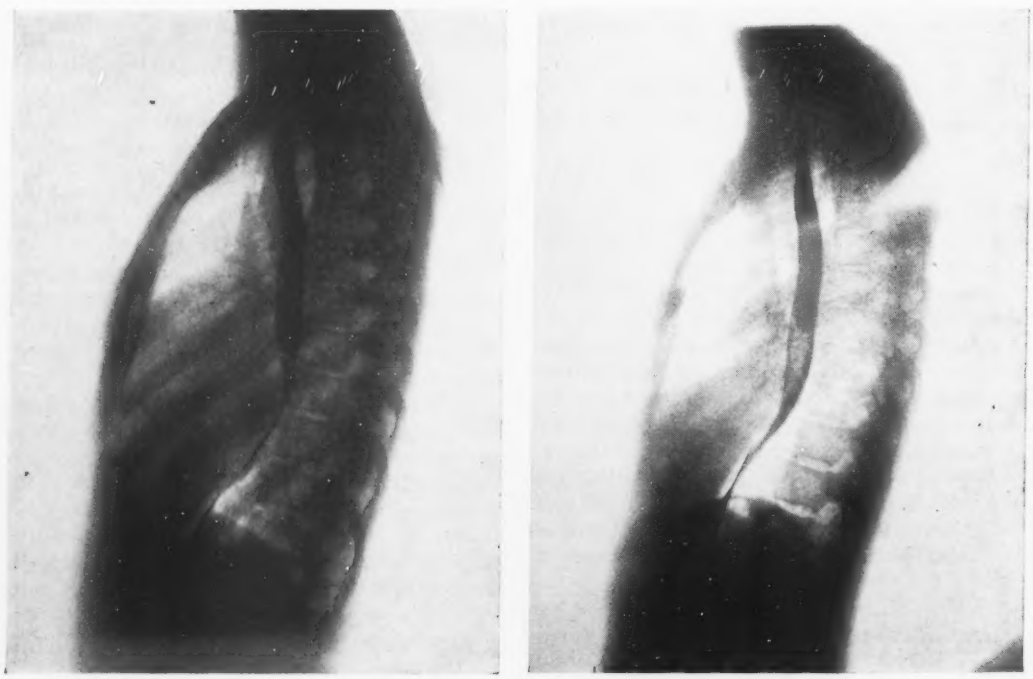

Figure 1

A B

Lateral x-rays of chest showing diminution in size of left atrium as outlined by barium swallow following mitral commissurotomy (B) as compared with pre-operative film (A).

pounds at the time of delivery. During this fourth pregnancy she had had several episodes of hemoptysis. She and her husband were eager to have another child.

At the time of admission she was experiencing vague lower abdominal and flank pains and was bleeding vaginally. She exhibited a Grade II low-pitched, soft, rumbling, diastolic murmur over the apical and mitral areas. Blood pressure was 90/60. The second pulmonic sound was accentuated. Electrocardiogram revealed digitalis effect, flutter-fibrillation, and right heart hypertrophy. Fluoroscopy and x-ray revealed an increased transverse diameter of the heart with definite enlargement of the left atrium (Figure 1A).

With the threatened abortion controlled, decision was made to perform mitral commissurotomy rather than to permit continuance of the complicated pregnancy or to perform a therapeutic abortion. The operation was performed on August 22, 1953. At operation, numerous pleural adhesions were encountered. The pericardial sac was unusually tense, and incision into the pericardial sac released a moderate amount of fluid. The enlarged left atrium of the fibrillating heart was entered through its auricle. Digital exploration revealed soft pliable valve leaflets, no calcification being present, with the valvular orifice measuring approximately 0.5 cm. in diameter. The anterolateral commissure was finger-fractured with only moderate difficulty, and the postero-medial commissure was easily split by a similar maneuver, resulting in an orifice which the operator (A.M.T.) estimated would admit 1½ to 2 fingers. No regurgitation was noted prior to or following the commissurotomy. The left auricular appendage was ligated at its base, partially amputated, and the open end oversewn with continuous fine silk suture. The amputated auricular myocardium showed mild fibrosis with no evidence of Aschoff bodies. A lung biopsy revealed only fibrous pleuritis.

The patient withstood the operation well. However, her convalescence postoperatively was prolonged. Her only serious postoperative complication was an atelectasis of the medial segment of the right middle lobe, which responded to bronchoscopic aspiration. She continued to receive digitalis and was discharged from the hospital on September 6, 1953.

During the remainder of her pregnancy her cardiac status did not become worse; in fact both dyspnea and edema were less than when examined by one of us (E.W.C.) in May 1953, prior to pregnancy. The prenatal course was uneventful.

She was delivered January 31, 1954, after a five-hour labor, of a living female infant weighing 4 pounds and 8½ ounces. Labor was spontaneous with a midline episiotomy. During labor 60 mgm. of nisentil was given and a pudendal block was done for delivery. During labor, delivery, and immediately postpartum, no signs of cardiac decompensation were noted.

Since delivery her weight has varied from 82 to 89 pounds. The severe cramping of her legs and coldness of her feet which were always present on exertion prior to operation have disappeared completely. Although she continued digitalis, she no longer finds it necessary to remain on a low salt diet. Edema of feet and ankles, which had been present prior to operation, is no longer observed. She is less susceptible to upper respiratory infections. Her exertional tolerance has improved to a point where only hard work tires her. She is able to do all of her own housework, including such tasks as waxing floors, without difficulty. She states that she can walk at a normal rate as far as desired without dyspnea. The mitral murmur, though still present, has been significantly diminished. An electrocardiogram still shows auricular fibrillation with a slow ventricular response. Her blood pressure remains unchanged.

Roentgenological examination reveals evidence of postoperative improvement. The transverse diameter of the heart is still enlarged, but there is some diminution in the size of the left atrium (Figure 1B).

DISCUSSION

The hemodynamic burden of pregnancy, as well as the natural course of rheumatic heart disease, accounts for cardiac deaths during pregnancy. Medical and surgical therapeutic means are available which control many of the factors giving rise to this hemodynamic burden.

These patients must be carefully and frequently observed. The curve of cardiac work in pregnancy, maximum in the seventh and eighth months, must be kept in mind. Often rigid cardiac regimens must be enforced, especially during the critical periods of pregnancy. The major principle is to organize ways and means which limit or restrict the rise in cardiac output, cardiac rate, and total blood volume. Limitation of physical activity, avoidance of emotional stress, proper attention to ectopic rhythms and tachycardia, digitalization, limitation of sodium intake, the avoidance of infusions or transfusions which may increase the blood volume, and proper attention to other factors (such as anemia, obesity, infections, and hyperthyroidism) are necessary. The severity of the heart disease, the age of the patient, the history or occurrence of heart failure, and the possibility of adequate supervision and accurate management must also be taken into consideration. It is generally felt that operation is not indicated in patients falling into functional classes 1 and 2.[9]

In cases in which the mitral valve is so tight and the patient's cardiac reserve so severely compromised

that no amount of supportive therapy will compensate for the load of pregnancy (Classes 3 and 4 patients), interruption of pregnancy or mitral valvulotomy must be considered. The demonstrated effectiveness and low mortality of this surgical procedure should give it priority over interruption of pregnancy. Patients with a history of doing badly from a cardiac standpoint during previous pregnancies, or who develop pulmonary edema in spite of adequate medical treatment early in pregnancy, should be offered this operation.

According to a recent report covering a period of 15 years, therapeutic abortions have varied from one abortion per 166 deliveries to one abortion per 357 deliveries in the average teaching hospital.[19] Cardiac cases account for 15 to 20 per cent. Approximately 85 per cent of these abortions for cardiac disease were in patients with pure mitral stenosis or combined lesions due to rheumatic infection.[19] Glover et al.[9] postulate that since the number of therapeutic abortions for tuberculosis has decreased, while the number for cardiac disease has remained nearly the same, this percentage in recent years has doubled in proportion, making it 30 to 40 per cent of all cases. In their recently reported series of 500 mitral commissurotomies, 1 per cent of the cases were operated upon during pregnancy without mortality and with improvement in all.[9] These patients would ordinarily have been considered for therapeutic abortion.

Proper judgment in selection or rejection of cases for surgery requires an understanding of the pathophysiological mechanisms of the cardiac disease and the physiological mechanisms of pregnancy, individually and combined.

PHYSIOLOGICAL CHANGES DURING PREGNANCY

Certain physiologic changes have been shown to take place in pregnancy, a knowledge of which is important in evaluating and managing the cardiac patient who is pregnant.

1. Oxygen consumption progressively increases during pregnancy to a maximum of 25 per cent above the non-pregnant state.[21] This results from increased demands of the pregnant uterus and fetus.[7, 26]

2. There is an increase in the respiratory ventilation volume relatively greater than the increase in oxygen consumption resulting in a lowering of alveolar and blood CO_2 partial pressures.[7]

3. Cardiac rate, pulse pressure, and venous pressure (in the legs) are increased during normal pregnancy.[26]

4. Total blood volume increases up to 50 per cent during pregnancy, a fall in hematocrit resulting from a relatively greater increase of plasma volume than red cell volume. The volume increases up to about the eighth month, and then decreases as term approaches.

5. Cardiac output increases to a maximum of 50 per cent in about the 32nd week, being achieved by both an increased heart rate and stroke volume.

6. The placental site contains large arteriovenous communications which add further to the increased cardiac load.

7. Labor itself adds considerably to the cardiac load by increasing oxygen consumption and cardiac output.

8. Cardiac decompensation occasionally occurs 24 to 72 hours following delivery, a phenomenon which has been attributed by some authors[5] to interruption of the placental circulation and occluding the "shunt" and allowing a transient increase in circulating blood volume.

PATHOPHYSIOLOGICAL MECHANISM OF MITRAL STENOSIS

Mitral stenosis produces changes in the circulation due to obstruction of the flow of blood through the reduced mitral valve orifice. Left atrial pressure rises, and, in the less severe cases, a normal cardiac output may be maintained at rest, but often not during activity. The elevated pressure in the left atrium is necessarily followed by a corresponding rise in pulmonary capillary pressure. In severe mitral stenosis, the diastolic time is not sufficient to permit adequate filling of the left ventricle, and an increase in heart rate further reduces this diastolic time. Thus cardiac output is further reduced, requiring a further rise in the atrial pressure. This vicious cycle is reflected in an elevated pressure in the pulmonary artery, especially during exercise, which may be demonstrable roentgenographically and determined by cardiac catheterization.

COMBINED EFFECTS OF PREGNANCY AND MITRAL STENOSIS

In all pregnant women there is an encroachment on cardiac reserve.[14] Pregnancy imposes an extra strain on an already struggling heart, causing a decreased margin of cardiac reserve and increasing the threat of decompensation. Severe pulmonary congestion, large hemoptyses, attacks of pulmonary edema, and cardiac decompensation are likely to develop.

The normal rise in cardiac output which accompanies pregnancy requires an increased rate of flow of blood through the stenotic valve, necessitating further increase in left atrial pressure. This is reflected in an increased pulmonary capillary pressure. This is further complicated by the elevation of atrial and capillary pressures resulting from the increased blood volume and tachycardia of pregnancy. Thus failure of cardiac output and pulmonary edema are constant

threats to the pregnant patient with mitral stenosis.

In some patients with mitral stenosis, aggravation of dyspnea occurs unexpectedly early—within a few weeks of conception. It has been suggested that this increase in dyspnea may be similar to that commonly noticed in women during the time of menses, and it may be related to fluid retention by hormonal control.[16]

It is obvious, therefore, that the physician concerned with these patients must take into consideration all factors—those due to pregnancy, those due to valvular disease, the combined effects of these two, and those changes due to other factors (obesity, anemia, etc.)—in making the best decision for these patients to permit attainment of the obstetrical ideal, a living mother and a living baby, without subjecting both to the dangers of a possibly needless operation.

ARGUMENTS AGAINST OPERATION DURING PREGNANCY

The history of mitral surgery, especially during pregnancy, is recent and requires further evaluation. The normal pregnant woman may exhibit many findings which mimic organic heart disease:[9, 11] arrhythmias (particularly paroxysmal auricular tachycardia), functional murmurs (heard in 50 to 70 per cent of all pregnant women), increased heart rate, breathlessness, nocturnal dyspnea, orthopnea, and ankle edema (due to venous congestion of the lower extremities). Intrathoracic changes during pregnancy, especially those resulting from elevation of the diaphragm and the lordotic posture of the patient, are suggestive of organic heart disease. These include an increased haziness of the lung bases (suggestive of pulmonary congestion), an illusion of cardiac enlargement, accentuation of the pulmonary conus,[20] and bulging of the upper segment of the left cardiac border with retrodisplacement of the esophagus[13] (mimicking the findings produced by enlargement of the left atrium seen in mitral stenosis). Murmurs that are impressive during pregnancy may, upon postpartum reappraisal, be minimal or even absent.

If the pathological state can be tolerated during pregnancy without too great risk, surgery may prove unnecessary. Pregnancy does not seem to hasten or adversely affect the pathological changes of rheumatic heart disease.[12] Women with mitral stenosis and pregnancy on the whole do well with informed medical treatment. While surgery may be performed successfully and the pregnancy continued to a successful conclusion, most of these women would probably do well without an operation and with adequate medical supervision.

Operative hazards are increased by the combined effects of pregnancy and cardiac pathology. Commissurotomy may reactivate rheumatic fever. Operation, if indicated upon completion of pregnancy, subjects only one life to the risk of surgery. Diagnosis, even in the best of hands, is uncertain. Regurgitation or multiple valvular disease may exist, and these patients may be subjected to operation without benefit and with increased risk both during and following cardiotomy. The operation does carry some mortality,[10] and even patients with pure mitral stenosis may be made worse by surgery.

The life of the fetus, in particular, is theoretically endangered by operation.[2] Bouts of cyanosis may occur during the induction of anesthesia or during the surgical maneuvers, which may also give rise to severe hypotension. This hypotension may be temporary or may persist postoperatively for varying periods of time. The fetal arterial blood is believed to maintain the same oxygen tension as the mother's venous blood. Therefore, even slight degrees of hypoxia in the mother's circulation may be reflected in a low venous oxygen tension and consequent extreme hypoxia of the fetus. Death of the fetus and subsequent abortion or miscarriage may result.

ARGUMENTS FOR OPERATION DURING PREGNANCY

Mitral surgery has made childbearing possible for many women heretofore doomed to barren marriage by severe rheumatic disease. Frequently marriages fail to survive because of this factor alone. Even in pregnancy, the results of the operation have been reported generally as good.[2, 9] Many feel the policy of terminating pregnancy and sterilizing these patients is no longer justified until the valve is actually examined at operation.[4, 7, 9]

Modern cardiology is not limited to clinical, radiological, and laboratory examination. It extends into the operating room where the final, more complete, and more exact assessment can often be made.[4, 9] Patients have been subjected to mitral commissurotomy after the pregnancy has been terminated and even after sterilization. Yet experience has shown that the risk of operation during pregnancy is an acceptable one.[9, 25] During pregnancy the lives of the patient and the unborn child are often in peril, and aggravation of symptoms such as dyspnea or acute pulmonary edema may occur unexpectedly early.

The operation may be performed at any time, even during the period of greatest hemodynamic strain, but it is generally agreed that early operation is preferable.[9] The earlier in pregnancy operation is undertaken, the sooner benefits accrue before the circulatory burden asserts itself. And, in the event of surgical failure, there will be more opportunity to resort to interruption of pregnancy—when this proves absolutely necessary.

The hormones of pregnancy may have a protective effect against reactivation of rheumatic disease fol-

lowing commissurotomy during pregnancy.[9] It is our opinion that abortion is less likely to occur after successful operation. Even if the pregnancy is completed successfully without operation, the mother may be left a cardiac cripple, in need of operation, unwilling or unable to leave the care of her child to undergo surgery, and yet less able to care for the child and home because of her cardiac disability. During this time, the cardiac condition in the more severe cases may undergo considerable progressive deterioration.

CONCLUSIONS

Every case of mitral stenosis during pregnancy must be carefully individualized. Supportive therapy, mitral commissurotomy, and occasionally therapeutic abortion may have a place in the treatment of these patients. Ordinarily only patients in Classes III and IV will require mitral commissurotomy.[9]

If, under proper medical care, signs of cardiac decompensation prior to the "safe interlude" preceding delivery do not develop, the outlook is relatively good with improvement in cardiac symptoms during the final weeks of pregnancy. The incidence of heart failure steadily increases in pregnancy up to the 32nd week, but primary failure seldom occurs after that time.[24] Labor is usually well tolerated and does not impose an unbearable strain on the heart.

However, in those patients who, in spite of intelligent medical management, develop stubborn congestive phenomena or give promise of doing so (history of doing badly in previous pregnancies in spite of good medical management, poor cardiac status prior to pregnancy, etc.), mitral commissurotomy is often preferable to therapeutic abortion.

Although mitral commissurotomy may be performed at almost any time, even during the period of greatest hemodynamic strain, the earlier in pregnancy it is performed, the better. The cardiac reserve diminishes as the burden of pregnancy increases, and the hazards of operation are progressively greater and its benefits less marked as pregnancy advances. Operation can best be done during the first trimester. The findings of patients in the 16th to the 32nd weeks of pregnancy should be carefully weighed before the operation is offered. It is contraindicated, as a rule, after the 32nd week.[9]

It is the opinion of many, including ourselves, that repeated embolic phenomena call for emergency mitral commissurotomy at any stage in pregnancy.[9]

Mitral commissurotomy is a diagnostic as well as a therapeutic procedure which should be utilized before termination of pregnancy.[4] Cesarean section or therapeutic abortion is, as a rule, seldom justifiable prior to operation. In many cases improvement will nullify the necessity of terminating the pregnancy.

On the other hand, if cardiotomy reveals a rigid, calcified mitral valve, moderate regurgitation or a high degree of cardiac enlargement, or if the therapeutic response following commissurotomy is poor (especially in patients over 35 years of age or in those with auricular fibrillation), termination of pregnancy may be justified, in spite of the performance of a technically adequate commissurotomy.[9] This is also true if other serious pathology is discovered at operation. Thus the findings at operation will enable the cardiologist and the obstetrician to determine whether or not the present and possible future pregnancies are too great a threat to the patient's life.

Alfred M. Tocker, M.D.
305 KFH Building
Wichita, Kansas

Ernest W. Crow, M.D.
2721 Boulevard Plaza
Wichita 16, Kansas

Grant E. Evans, M.D.
435 North Hillside
Wichita 6, Kansas

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Rectal and Colonic Cancer

with Special Reference to the Importance of Precursory Lesions

CHARLES W. MAYO, M.D., *and* DONALD R. DAVIS, M.D.,
Rochester, Minnesota

When it is considered that 15 per cent of malignant tumors of the human body occur in the anus, rectum, and colon, that 30,000 to 35,000 American people die annually of this disease, and that approximately 12 per cent of persons harbor lesions of malignant potentialities in the colon or rectum at some time in their lifetime, impetus is given to efforts advanced for the eradication of precancerous lesions in this region.

We are in agreement with Fansler[1] in his belief that further extension of the surgical attack on cancer of the colon and rectum will offer little in the way of improving our present statistics on salvage. Furthermore, we are of the opinion that greatest improvement will evolve from efforts to detect and eradicate certain premalignant lesions, namely adenomatous polyps of the rectum and colon, familial polyposis, and chronic ulcerative colitis. Thus, it is apparent that the task of improving our present salvage rate is not one that falls only to the skill of the surgeon but, rather, is the prerogative of all physicians actively engaged in the practice of medicine.

It is generally agreed that adenomatous polyps of the colon and rectum constitute one of the most dangerous precursors of cancer known,[2] and furthermore, this precursor is the most common. In a recent report, Hitchcock and Aust[3] reviewed the records of 5,352 men and women examined at the Cancer Detection Center of the University of Minnesota between March, 1948, and February, 1953. They found that 1.7 per cent of these patients were hosts to clinical cancer and that the colon and rectum were the primary sites of the lesions twice as frequently as was any other organ. Thirteen hundred patients, or 24 per cent of their total group, had precancerous lesions of one form or another. Six hundred twenty-four of these lesions, or approximately half, were adenomas of the rectum or colon. This represents an incidence of 11.7 per cent of all persons examined. It is our opinion that all these people, if given sufficient time, ultimately would have clinical cancer and that a portion of them would die of the disease.

Improvement in cure rates for cancer of the colon and rectum is more likely to result from proper treatment of precancerous lesions than from extensions of our operative procedures. Conditions considered include adenomatous polyps, familial polyposis, and chronic ulcerative colitis.

Whether or not all cancers of the colon and rectum have their origin in adenomatous polyps has been a much-debated question and, of course, the answer is not known. Jackman and Mayo[4] outlined the evidence that has accumulated in recent years to indicate that polyps of the colon and rectum are important forerunners of cancer. This evidence, we feel, is worthy of reiteration, as follows: (1) There is a close parallelism between the location of polyps and the location of clinical cancer in the large intestine; (2) There also is significant similarity of age and sex distribution between patients who harbor adenomas and those

Dr. Mayo is affiliated with the Section of Surgery, Mayo Clinic and Mayo Foundation, and Dr. Davis, who is now practicing in Mission, was a Fellow in Surgery, Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the graduate school of the University of Minnesota.

who harbor carcinomas; (3) The frequency with which adenomas and carcinomas are encountered in the same patient is noteworthy; (4) Persons with familial polyposis almost invariably die of cancer of the colon or rectum if they are not treated and if they live long enough; (5) Untreated polyps show malignant degeneration on subsequent examination; and (6) Polypoid disease and subsequent carcinoma are among the most common complications of chronic ulcerative colitis.

Seventy per cent of cancers that involve the large intestine are within reach of the 24-cm. sigmoidoscope. The same is true of adenomatous polyps. Cancer of the colon occurs most frequently in patients between the ages of 30 and 70 years, the so-called cancer age. Again, the same is true of adenomatous polyp. Both adenomas and carcinomas predominate in men in a ratio of 3:2. Eighteen per cent of specimens removed because of carcinoma of the colon have one or more polyps adjacent to the growth, which are commonly designated "sentinel polyps." If it is considered that the remainder of the colon may harbor tiny polyps not visible by roentgenographic means, the actual incidence must be a good deal higher than this.

Buie[5] has estimated that 25 to 30 per cent of all persons with cancer of the colon or rectum have associated polyps somewhere in the colon, but he has stated that the majority occur close to the growth. Dockerty[6] is of the opinion that approximately 50 per cent of persons with familial polyposis will die of cancer by the age of 30 years, whereas practically all of them will have advanced abdominal cancer or will die of cancer before the age of 50 years. Buie and Brust[7] reported on eight patients who had solitary rectal adenomas and refused treatment. In four of these cases, clinical cancer developed at the site of the original polyps during periods ranging from 2 to 10 years. Bacon and associates[8] studied 800 cases of cancer of the colon and reported that in 90, or 11.2 per cent, there was evidence that the carcinoma had its inception in an adenomatous polyp, and that in an additional 3.7 per cent there was presumptive evidence of such origin. A patient treated by us illustrates vividly the adenoma-carcinoma sequence.

Illustrative Case. A machine operator, aged 52 years, first came to the Mayo Clinic in 1948 because of nervousness and worry about his health. After a satisfactory examination, including digital rectal examination, he was dismissed with a diagnosis of anxiety neurosis. In 1952 he experienced an episode of rectal bleeding and consulted his home physician; proctosigmoidoscopic examination revealed two small polyps in his rectum. Immediate treatment by fulguration was recommended but was refused by the patient. In September, 1954, after two years of procrastination, he was referred to this clinic by his home physician for treatment of rectal carcinoma. On proctoscopic examination, the diagnosis of annular, ulcerative, obstructive adenocarcinoma, grade 2 (Broders' classification), was confirmed. An operation that was undertaken with hope for a cure revealed extensive hepatic metastasis which involved both lobes of the liver, so only a palliative resection was performed to relieve intestinal obstruction.

Here, then, was a man doomed to early death because of his procrastination in the treatment of rectal polyps. It may seem redundant to emphasize that patients with rectal polyps who delay treatment should be fully informed of the hazard of cancer and, should they persist in refusing treatment, the responsibility should be entirely their own.

Twenty-five years ago it was not believed that cancer ever occurred as a complication of chronic ulcerative colitis. However, since the report of Yoeman,[9] in 1927, there has been an ever-increasing number of reports in the medical literature connoting its frequency. Bargen and associates[10] recently reported 1,564 cases of chronic ulcerative colitis and attempted to determine the relative frequence with which carcinoma occurred as a complication. They determined that 6.3 per cent of patients in their group died of carcinoma of the colon. Not concerning themselves with the number of cases in which cancer may develop later, they compared the number of deaths from cancer in their group with the expected number of deaths from cancer of the colon in the normal population, utilizing death rates supplied by the United States Public Health Service. They concluded that cancer of the colon and rectum develops 30 times as frequently in persons who have chronic ulcerative colitis as it does in individuals of similar age groups in the general population.

It has been noted that the incidence of malignancy is greater in persons afflicted with chronic ulcerative colitis at an early age; that in this group of patients the grade of malignancy usually is high; that the lesions frequently are multiple, and that the outlook for these patients is extremely grave. Of 73 patients studied by Shands and associates[11] and subjected to operation with hope for cure, only two lived for five years and one of these subsequently succumbed to carcinoma.

Having presented evidence illustrating the serious danger of cancer associated with adenomatous polyps, familial polyposis, and chronic ulcerative colitis, we are confronted with the problem of how we might deal with these conditions to advantage from the aspect of prevention of cancer.

ADENOMATOUS POLYPS

It is fundamental that physicians and surgeons should be aware of the high incidence of polyps in seemingly normal persons. In evaluating the advantage

of routine proctosigmoidoscopy, Hauch and associates[12] reviewed the records of 1,919 patients who had had no symptoms referable to the anus or gastrointestinal tract. They found that 8.1 per cent of this group had adenomas of the rectum or sigmoid colon. Baggenstoss,[13] in a study of 2,784 necropsies in which death was attributable to diseases not related to the colon, found that 14.7 per cent of the men and 9.0 per cent of the women had polyps in the large intestine. In view of the fact that one in 10 patients more than 40 years of age has polyps that are visible through the 24-cm. sigmoidoscope, the "polyp-conscious physician" can render a great service to his patients by finding and destroying these lesions in their early stages of development.

Medical schools, hospitals, and teaching clinics are urged to familiarize students with the proper handling of proctosigmoidoscopic equipment and to teach with emphasis the value of adequate examination of the rectum and terminal portion of the colon. Practitioners not fully acquainted with the technic of proctosigmoidoscopic examination should take time off from their busy practices to observe and learn this technic so that they may render this service to their patients. We feel that every patient who undergoes a routine examination and is 40 years old, or older, should be subjected to proctosigmoidoscopic examination and, perhaps, to x-ray examination of the colon.

Once a polyp has been discovered, its immediate removal is mandatory. The size of the growth gives no indication as to whether or not malignant change has taken place, nor can it be predicted when a given polyp will undergo malignant change. It is not possible to tell, by gross inspection, which polyps are benign and which are malignant; microscopic study is necessary to determine their true nature. Once a polyp has been discovered and treated, either by fulguration or by surgical removal, the patient should be instructed to watch carefully for signs of rectal bleeding. Also, he should have a proctoscopic examination and barium-enema study at least once a year thereafter. It has been shown that in approximately 20 per cent of cases, additional polyps develop after fulguration of rectal polyps, and that in 30 per cent of cases additional polyps develop after transcolonic polypectomy. The necessity for careful follow-up is therefore obvious.

FAMILIAL POLYPOSIS

Total abdominal colectomy is the only safe treatment for familial polyposis, in view of its serious malignant potentialities. This view is universally accepted. In general, we follow one of two approaches to the problem, depending on the presence or absence of cancer of the rectum.

In the presence of cancer of the rectum, we prefer to carry out total abdominoperineal resection with establishment of a permanent ileac stoma. The node-bearing tissues along the superior hemorrhoidal and inferior mesenteric vessels are removed en bloc, as in any other adequate operation for cancer. We prefer either skin-grafting the ileac stoma or cuffing it with mucous membrane, believing they offer certain advantages to the patient.

In the absence of cancer of the rectum, provided the rectum is not diffusely and completely involved with polyps, total abdominal colectomy with preservation of the rectum and ileorectostomy is the operation of choice. Polyps in the rectal remnant are fulgurated through the sigmoidoscope prior to or at a propitious time after the operation. We prefer one-stage resection with primary end-to-end anastomosis because of certain technical advantages to both the patient and the surgeon, as well as great economic saving to the patient.

Follow-up examination of the rectum in the years after operation is extremely important and must be emphasized to the patient; we advise it be done every six months. In practically 100 per cent of patients additional polyps develop, and these will require fulguration. Black and Hansboro[14] found that, of 26 patients, all but one had polyps in the rectum that required fulguration each year for four years after operation. Total colectomy with ileorectostomy will be successful only if the polyp-bearing rectal remnant is examined periodically and indefinitely.

CHRONIC ULCERATIVE COLITIS

Because cancer develops 30 times as frequently in patients with long-standing chronic ulcerative colitis as in persons not so afflicted, it behooves the attending physician to observe these patients carefully for polypoid change and for symptoms suggestive of supervening malignancy. Opinion is widely divided as to the proper method of dealing with this condition. There are those who advocate total colectomy with permanent ileac stoma in every case as soon as the diagnosis has been established definitely. On the other hand, others adhere to a more conservative view, resorting to operation only in the event of definite indications, such as severe intractability, hemorrhage, polypoid change, or malignant degeneration.

In general, the problem of selecting patients for operation has not been standardized. In view of the great number of patients who have been treated successfully by medical means alone, we are convinced that routine total colectomy with ileostomy borders on the unjust and that surgeons should maintain an open mind, weighing carefully the question as to whether surgical treatment is necessary to return the patient to a useful life. We have leaned toward the conservative school in the treatment of chronic ulcerative colitis, but we feel that all patients, with or without ileostomy, should be examined periodically, with re-

view of their histories and with laboratory studies, proctoscopic examination, and barium-enema study of the colon to detect any supervening malignancy at the earliest possible moment. In reviewing the management of this disease at the Mayo Clinic, we found that total colectomy has been performed three times more frequently for chronic ulcerative colitis after 1940 than it was prior to 1940. We do total colectomy in approximately 12 per cent of cases at present.

With reference to polyps complicating chronic ulcerative colitis, those within reach of the sigmoidoscope should be fulgurated, preferably during a remission to avoid flare-up of the disease, and those above the proctoscopic level should be checked frequently by roentgenologic and fluoroscopic examinations to detect any change in size. Should a polyp show enlargement on successive examinations, the intervention of malignancy should be seriously considered and the patient should undergo total colectomy.

SUMMARY AND CONCLUSIONS

1. Early detection and eradication of precancerous lesions rather than further extension of our operative attack will improve our present salvage rates in carcinoma of the colon and rectum.

2. Adenomatous polyps in the colon and rectum are important forerunners of cancer and should be removed. One person in 10 past the age of 40 will have polyps visible through the 24-cm. sigmoidoscope.

3. Total abdominal colectomy is the only safe treatment for familial polyposis, and successful surgical treatment after ileoproctostomy hinges upon adequate follow-up and treatment of the rectal remnant.

4. Persons with chronic ulcerative colitis have cancer of the colon and rectum 30 times as frequently as do persons not so afflicted. Periodic review of the patient's history, physical examination, laboratory studies, proctoscopic examination, and barium-enema studies will determine the question of medical or surgical therapy.

5. A plea is made for medical schools, hospitals and teaching clinics to familiarize their students with the proper use of proctoscopic equipment. Busy practitioners should take time away from their practices, if need be, to learn how to perform an adequate proctosigmoidoscopic examination.

Charles W. Mayo, M. D.
Mayo Clinic
Rochester, Minnesota

Donald R. Davis, M.D.
5830 Nall Avenue
Mission, Kansas

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People sometimes refer to higher education as the higher learning, but colleges and universities are much more than knowledge factories; they are testaments to man's perennial struggle to make a better world for himself, his children and his children's children. This, indeed, is their sovereign purpose. They are great fortifications against ignorance and irrationality; but they are more than places of the higher learning—they are centers and symbols of man's higher yearning.

—*W. H. Cowley*

Rheumatic Heart Disease

The Experimental Production of Rheumatic-Like Lesions in the Hearts of Animals

VIRGINIA LEE ENGLAND TUCKER, ***Kansas City***

INTRODUCTION

Reproduction of characteristic heart lesions of rheumatic fever in experimental animals has been the subject of much research and speculation particularly concentrated in the past 25 to 30 years. Demonstration of the heart lesions of rheumatic fever in experimental animals is essential to the establishment of etiological factors and the treatment of the disease. Outranked only by tuberculosis and syphilis, rheumatic fever is the third most common serious infectious disease in this country. It is the foremost cause of death in individuals below the age of 20.[5] Before measures of prevention and control can be established, an experimental model must be reproduced.

Historically the definitive signs of the disease have been observed with sufficient frequency to enable some basis for judgment and analysis of experimental results.

In 1805 J. Haygarth[8] led the research in the field by the observation that clinically these lesions were a "cyanche, a not uncommon antecedent of attacks of rheumatic fever."

The microscopic requirements of the lesions in the acute phase of the disease were described by L. Aschoff[1] in 1904. His classic description of Aschoff bodies as specific lesions of rheumatic fever has been the measuring device for the production of the disease in the heart experimentally.

An attempt to formulate the lesions demonstrated in the heart to a pathogenesis of a disease process was made by Shick[25] in 1907. The essence of his suggestions related the endocarditis to an "eminent allergic sequelae" of rheumatic fever.

The etiologic factor of the disease was defined by A. F. Coburn[3] in 1931. A hemolytic streptococcus was considered to be implicated in epidemiological and bacteriological studies of rheumatic fever patients.

Rebecca Lancefield[13] did much to restrict the etiological agent to a specific organism by demonstrating that Group A streptococcus was serologically specific for man.

The purpose of this paper is two-fold: (1) to review the experimental work which has been done in the production of the heart lesions characteristic of rheumatic fever, (2) to analyze the positive and negative findings in experimental animals simulating the heart lesions of rheumatic fever. A complete review of the literature has not been made. It is felt, however, that an adequate sampling of the representative types of experimental work sufficient to illustrate the major trends on the subject of the paper has been accomplished.

REVIEW OF EXPERIMENTAL WORK

The experimental work which has been done in the production of rheumatic heart disease in animals can arbitrarily be placed in five categories based on theories pursued by investigators concerning the pathogenesis of rheumatic fever. They are as follows: (1) a hypersensitive reaction to hemolytic streptococcus, (2) the effects of trauma or stress placed on the heart with a superimposed streptococcal infection, (3) the effects of nutritional deficiency with a superimposed streptococcal infection, (4) the synergistic effects of streptococcus and vaccinia virus, and (5) an immune reaction based on a parasite-host response mechanism.

Before an experimental research animal can be said to demonstrate the changes and cardiac lesions of rheumatic fever, the complete course of the disease should be observed, not just the more obvious initial stage. In order to accomplish this, the experiment must be maintained for long enough periods of time that animals can demonstrate these lesions.

The first category concerns experimental work based on the assumption that lesions found in the hearts of rheumatic fever patients result from a hypersensitive reaction.

McKeown[15] induced a hypersensitive response in the hearts of rabbits by repeated injections of horse serum. Microscopically, the tissue response to sensitization by horse serum was evidenced by focal necrosis of collagen in perivascular tissue, interstitial septum of the myocardium, and fibrous tissue formation on the valve rings and valves. The inflammatory re-

Mrs. Tucker is a student at the University of Kansas School of Medicine. This paper was written during her sophomore year at the school.

sponse of the myocardium and perivascular region was felt to simulate Aschoff nodules.

Kirschner and Howie,[12] using rabbits, repeated the work of McKeown. However, they enlarged the experiment to include not only the hypersensitive response to horse serum but also any apparent synergistic effects that might be present from multiple injections of horse serum and killed streptococci. A third group of rabbits was given multiple injections of Group A hemolytic streptococci. Cardiac lesions were observed to vary only in degree of severity in cases where streptococci were used. Incidence of lesions within a group was approximately the same in all three groups of rabbits.

Robinson[23] used both monkeys and rabbits, but soon abandoned monkeys since only mild carditis could be produced. The intent of his experimental work was to demonstrate the cardiac lesions characteristic of rheumatic fever were related to a hypersensitive response rather than to an infectious process. He made comparative studies of groups of rabbits given repeated inoculations of Group A streptococci, single inoculations of streptococci, multiple injections of diphtheroids, heated mouth washings of rheumatic patients, and unheated mouth washings of rheumatic fever patients. The most severe cardiac lesions were produced in rabbits subjected to multiple injections of Group A streptococci. Although he had no evidence of such experimentally, he felt that the factor responsible for the lesions was a "heat-labile, nonantigenic factor, a toxic albumin—bacterioplasma," a specific toxin of streptococci.

Murphy and Swift[19] did a series of experiments over a two-year period attempting to produce a rheumatic fever-like state in rabbits by successive focal injections of heterologous types of Group A streptococcus. The rabbits were observed from three to 20 months before being sacrificed. The heterologous streptococcus under the circumstances of the experiment produced lesions more typical of human rheumatic fever than had previously been demonstrated.

Schultz and Fite[24] sensitized rabbits with an initial intravenous injection of acacia followed by multiple injections of serum derived from rheumatic fever patients. The animals were sacrificed in three months after approximately seven to 12 injections. Vascular lesions similar to those rather characteristically seen in cases of human rheumatic fever were observed. Other findings were regarded as equivocal.

Thomas and Stetson[28, 29, 30] induced a generalized Swartzman reaction in rabbits which resulted in valvular, perivascular, and myocardial lesions of the heart. Coronary vessels were observed to undergo fibrinoid necrosis. Deposits of fibrinoid occurred on the mitral and aortic valves, principally on valve surfaces. Affected valves displayed fragmentation of the ground substance. Often encountered in the vicinity of blood vessels were areas of necrotic myofibrils infiltrated by mononucleated inflammatory cells possessing basophilic cytoplasm and characteristic "owl-eyed" nuclei.

Instead of giving streptococci as the preparatory and provocative agent in producing the generalized Swartzman reaction, Thomas, Denny, and Floyd[31] produced similar lesions in the heart by using streptococci as the preparatory agent and meningococci as the provocative agent. No cardiac lesions were produced using meningococci alone.

The second category concerns experimental work based on the assumption that lesions may be produced by trauma or stress placed on the heart with a superimposed streptococcal infection.

Lillehei, Hammerstrom, Wargo, and Clawson[14] produced bacterial endocarditis by surgically producing A-V fistulas in dogs followed by intravenous injections of Group D hemolytic streptococci. A similar procedure was followed in a second group of dogs except that after septicemia had been established, a series of antibiotics was administered. In the six animals in the first group of dogs not receiving antibiotics, 100 per cent developed bacterial endocarditis. In the second group, dogs receiving antibiotics, endocarditis was produced in four of the five dogs tested. The mitral valve was most frequently involved.

By subjecting rats to the equivalent of an altitude of 25,000 feet, Highman and Altland[10] produced fibrinous nodules on the heart valves of rats. Having produced the initial lesions, they were able to produce bacterial endocarditis, particularly valvulitis on the mitral and aortic valves. They used a variety of organisms isolated from cases of human bacterial endocarditis.

The third category, the effects of nutritional deficiency with a superimposed streptococcal infection, was demonstrated by Rinehart and Mettier.[22] They were able to show a degenerative and proliferative valvular lesion in the hearts of scorbutic guinea pigs by infection with Group A hemolytic streptococcus. The limiting factor of this experiment is that similar lesions were evidenced with organisms other than streptococci.

The fourth category, the synergistic effects of streptolysin O and vaccinia virus, was observed incidentally in an experiment designed specifically for the induction of rheumatic and rheumatoid arthritis. Sokoloff and Beegel[26] noted a high frequency of "viral carditis" in rabbits receiving injections of streptolysin O and vaccinia virus. The lesions were characterized by myocarditis and acute mitral valvulitis.

The fifth category, an immune reaction, was proposed by Calvelti.[2] He attempted to demonstrate experimentally in rats that the lesions of rheumatic fever result from the ability of streptococcus to combine

with substances of heart tissue. The streptococcal-tissue substance union acted as an antigen, resulting in the production of auto-antibodies by the tissues to this antigen. The combination of this auto-antibody and antigen led to the damage of these tissues. Emulsions of heart tissue and connective tissue were used respectively with soluble streptococcal protein and ether killed streptococcus. The most typical lesions were produced by heart and connective tissue emulsions. Connective tissue involvement was characteristic of both cases.

RESULTS OF EXPERIMENTAL WORK

In all cases reviewed, endocarditis was produced. The question still remains, "Have the identical lesions of human rheumatic fever been duplicated experimentally?"

The time element in the development of rheumatic phenomena in the course of the disease is important. Three phases plus the possibility of a residium or sequelae to the disease are outlined by Colburn.[4] The first phase is associated with infection. Activity is concentrated on an exudative process with varying degrees of intensity. The second phase results in a specific histologic change in the endothelial cells. Collagen necrosis, fragmentation of myocardium, and deposition of fibrin and the trapped serum contents can be demonstrated microscopically. The appearance of the third phase may occur several weeks or months after the onset of the initial infection. A period of quiescence may supersede this phase in which no apparent infection is present. The manifestation of sequelae is obscure but appears in a sizable number of cases. The most striking differences of the reactivity of the patient during the progress of the disease are (1) the primary lesions being associated with infection, and (2) the late lesions being associated with disturbances in metabolism.

Gross and Ehrlich[6, 7] described the life cycle of the Aschoff body which appears during the early stage of the disease. Since the Aschoff body has been credited as the discrete histological evidence of the primary stage of rheumatic fever, it has been the center of much attention in experimental work on rheumatic fever. Aschoff bodies pass through an orderly sequence of changes ending in an "intrafollicular scar." They appear in sites where collagen is most abundant: "in planes between the myocardial bundles, in fibroelastic tissue around blood vessels, and in the endocardium." In consequence, the architecture of the Aschoff body is subject to much variation depending on the stage of the disease and the type of tissue in which it lies. Murphy[17] believes he has been able to demonstrate that the Aschoff body arises from injured myofibrils rather than directly from collagen.

Experimental findings which most closely resemble the lesions of rheumatic heart disease are centered on the infectious organism responsible for the primary infectious phase of the disease and the sequence of events which occur during the primary stage. It has not been possible to define the mechanisms or the pathogenesis of the phase of the disease concerned with the "non-suppurative post-streptococcal disorders"[21] experimentally.

The most typical and consistent results closely simulating the picture seen in rheumatic patients were demonstrated in those animals given repeated injections of the Lancefield Group A streptococcus. The use of heterologous types of Group A streptococcus by Murphy and Swift[19] duplicated human streptococcal infections which have been shown to vary as to serological groups at different times. Dr. Lowell A. Rantz[21] considered the work of Murphy and Swift,[16, 19] and Kirschner and Howie[12] to have fulfilled the requirements necessary to say that lesions of rheumatic heart disease have been produced. He said, "Anatomical study of the tissues of these animals (rabbits) revealed lesions indistinguishable from those seen in fatal rheumatic fever in adults. The most severe tissue lesions appear in those animals that responded to later injections of living streptococci by a very vigorous antibody production of the type seen in rheumatic fever in man." The fact that in the case of Murphy and Swift the animals were maintained for a period of three to 20 months certainly made an analysis of the total effects of the disease on the heart in sequential order simpler.

The generalized Swartzman reaction produced cellular thrombi in the myocardium, pericardium, and valves of the hearts of rabbits. Some investigators felt that these thrombi which have been shown to be of leukocyte-platelet origin are as pathognomonic of the disease in the heart as Aschoff bodies. These lesions were shown to be specific to Group A streptococcal infection.[27]

The results of elevation of rats to 25,000 feet[10] and the stress of inducing A-V fistulas[14] in dogs plus superimposing streptococcal infections revealed other mechanisms which are being studied, having been revised in attempts to produce the changes of rheumatic fever.* Bacterial endocarditis has been produced consistently, but the characteristic effects of rheumatic fever on the heart were not demonstrated experimentally.

Epidemiological studies of the incidence of rheumatic fever reveal that "rheumatic fever occurs most frequently in those sections of large cities in which the economic level is low and standards of living poor."[21] Credit for the recognition of increased inci-

*Hamilton, T. R.: Personal communication regarding research currently in progress.

dence of rheumatic fever among persons who would most likely be subjected to vitamin deficiencies can be given to Rinehart and Mettier[22] for their attempt to produce rheumatic heart disease with hemolytic streptococci in scorbutic guinea pigs. Environmental factors may have more significance than is recognized today.

Experimental data concerning lesions producing "viral carditis" in rabbits receiving injections of streptolysin O and vaccinia virus were not described sufficiently to enable discrete analysis.[26]

In animals in which Cavelti[2, 20] was able to produce heart lesions by injection of emulsions of heart or connective tissue with soluble streptococcal protein extracts, results indirectly indicated that an autoantibody has been produced. A time lag was noted before the lesions were produced. Lesions were present only when an autogenous tissue was used. No reaction in the heart was observed with skeletal muscle devoid of connective tissue. Serological studies were made in vitro with extracts of plain homologous tissue component antibodies demonstrable with serum from animals treated with either heart or connective tissue emulsion and killed streptococci.

DISCUSSION

The basis for evaluation of experimental data on rheumatic fever is placed solely on its contribution to a better understanding of the pathogenesis of the disease. Much progress has been made experimentally as to the infectious agent responsible for initiating the sequence of events. Experimental results have conclusively pointed to Lancefield Group A streptococcus. Why some individuals are susceptible to an attack of rheumatic fever and others are not under similar circumstances of repeated focal infections is unknown. Experimentally it was revealed that there was little consistency among animals concerning the degree of involvement of the heart. Some appeared more resistant than others, although the amount of exposure to infection was well controlled.

The significance of multiple infections or injections, as the case may be, appears significant both clinically and experimentally. In all cases of experimental success in producing some type of heart lesion, a sensitizer was required whether it be horse serum, a virus, trauma, or an initial dose of streptococcus. The logical conclusion would equate the disease to either a hypersensitive or immune reaction which requires a characteristic pattern of response to stimulation on the part of the host tissue.

The presence or absence of Aschoff bodies has been the cause of much debate among investigators at the experimental level. Murphy and Swift[18, 19] claim to have produced in rabbits the identical microscopic picture of Aschoff bodies seen in humans. Other workers have described swollen collagen fibers surrounded or infiltrated with "owl-eyed" cells. Hamilton and Syverton[9] have described the origin of these cells as tissue mast cells brought into play during the first phase of the disease. A host response to insult results in alteration of the appearance of these cells by "degranulation." Although experimental work is not conclusive that this is the case, they suggest that deficiency in this granular material may be important.

Gross and Ehrlich[6, 7] have outlined a life cycle of the cell which indicates that variation in appearance of the Aschoff body can exist in patients, depending on the stage of the disease at the time they are presented for autopsy. Group A streptococcus is a natural pathogen for man, not experimental animals. Therefore, tissue response may vary because of altered reactivity of animal tissue cells to a foreign pathogen, or to the stage of the lesion at which the observation was made. Aschoff[19] has emphasized the importance of applying the same etiological stimulus to different species of animals and comparing responses if one is to establish common cause for their responses.

Leukocyte-platelet thrombi in areas of Aschoff or Aschoff-like bodies and in coronary vessels were observed both on patients brought to autopsy with rheumatic heart disease and in experimental animals. Stetson and Watson[27, 32] demonstrated that the verrucae seen on the valve leaflets consist of "masses of blood platelets with unmeshed leukocytes, erythrocytes, and strands of fibrin" which appear much like "parietal thrombi." The suggestion is that the leukocyte-platelet thrombi and the verrucae on the valves are of common origin.

Experimental work done on animals has contributed significantly to the "phase 1 and phase 2" picture of rheumatic heart disease as outlined by Coburn.[4] Only until someone has demonstrated the complete gamut of the disease can the production or lack of production of the lesions of rheumatic fever experimentally in laboratory animals be analyzed. The most apparent fault of the experimental work that has been done is the impatience of research workers. The animals have either died or been sacrificed before the stage of quiescence has been bridged and the third phase or the sequelae of rheumatic fever have developed. Mitral stenosis, shortening of the chordae tendinae, dilatation of the left auricle with a mural thrombus climbing up its wall approximating the mitral valve, signs of decompensation, etc. are significant to the pathologist at the autopsy table in formulating the pathogenesis of rheumatic fever. In consequence, it appears to be a goal to be reached experimentally.

SUMMARY

Before the etiological factors essential to the pathogenesis of the heart lesions of rheumatic fever or treatment of the disease can be accomplished, an experimental model must be produced. A sampling of the significant attempts to reproduce the lesions of rheumatic heart disease experimentally has been reviewed. An effort to evaluate results of experimental work on the basis of what has been observed clinically and at the autopsy table has been made. A summary of this work follows:

1. Lancefield Group A streptococcus has been conclusively implicated as the initial factor in a sequence of events leading to rheumatic fever.

2. The necessity of multiple injections of Group A streptococcus before characteristic heart lesions can be produced is suggestive that the basic mechanism of the disease is related either to a hypersensitive or an antigen-antibody reaction.

3. The factor of biological variation enters into the picture plus the changes in the life cycle of the Aschoff body. These must be considered before success or failure of an experiment is asserted.

4. On the basis of experimental observation and postmortem pathological observation, leukocyte-platelet thrombi in coronary vessels and areas in close approximation to Aschoff bodies are similar to the verrucae seen on the mitral and aortic valves.

5. Experimental work has covered only the primary stage of the disease. Lesions of mitral stenosis and the sequelae of rheumatic fever have not been produced experimentally.

University of Kansas School of Medicine
39th and Rainbow
Kansas City 3, Kansas

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PRESIDENT'S PAGE

DEAR DOCTOR:

It is a strange anxiety you feel when you become president.

The responsibility weighs more heavily than you believed it would.

Problems seem larger and more formidable when you stand before them.

You are determined to uphold the high tradition of medicine, and realize, for the first time in your life, that a president simply cannot do this without help.

And you say you will ask for the cooperation of every member, and every auxiliary member, to accomplish the goals you have in mind:

Like the goal of achieving a better public understanding of what medicine has to offer, and what medicine stands for.

Like the goal of revising antiquated laws, so medicine may better serve the public.

Like the goal of improving patient care.

Then the thought strikes you that these have been the objectives of every president before you. They also asked for help, but you were sure they meant someone else.

Why didn't this occur to you years ago? Had you assumed more responsibility in the past as a physician and as a citizen, some of your present problems might have been solved.

So you experience a strange anxiety as you become president, and you wonder about your friends. Do they know how you feel, and how urgent these problems are? Will they help? I hope so.

Fraternally yours,

Clyde W. Miller M.D.

CLYDE W. MILLER, M.D.

EDITORIAL COMMENT

The Free Public School

Editor's Note. Although prepared as an address and not an editorial, the following paragraphs by Dr. Richard Greer, a member of our Editorial Board, seem destined for a wider audience than that which heard his talk, delivered at the dedication of the A. J. Stout Elementary School in Topeka on January 19. Dr. Greer's thoughts are reproduced here without his knowledge or consent.

In dedicating this school tonight to the memory of A. J. Stout, we do more than honor a man, we reaffirm an ideal. When we opened the doors of this building we opened them to all children regardless of their heritage, social position, or ability. This is the American Ideal—the Free Public School—a concept not known before this nation was founded, and even now seldom realized elsewhere. This ideal has led to troubles; the tax burden is great, school systems become large and unwieldy, teachers are scarce, and the results, to some critics, seem poor indeed.

I think that the record deserves some support. The specious arguments about teaching methods with the same turgid emotional reasoning are making headlines. Although we adhere to the philosophy that all children will be taught equally, we cannot guarantee that they will learn equally well. However, if we look at the staggering technical and scientific achievements of the past fifty years, we are struck with the fact that somebody learned something, somewhere. I admit, though only grudgingly, that we may lag in the humanities, and I am afraid that often we do not have time to dream, and if our dreams do reach for the stars they are likely to take the form of a skyscraper, a bridge, or a drill rig. I submit to you, that of course Johnny can read! But, I also insist that we need to devote our energies to content—to make wisdom our goal—that all our knowledge be applied to human good.

I think that it is nice to remember that this dedication is being made in memory of a dedicated man—a man dedicated to the American Dream—the Free Public School!

Medical History

Two significant projects designed to assemble and preserve the history of pioneer medicine in Kansas are now in operation. Both need the cooperation of individual Kansas physicians.

The Department of History at the University of Kansas is performing exhaustive research through newspaper archives in the preparation of a book entitled *A Century of Medicine in Kansas*. In addition, the Kansas Medical Society is making available out of its files the minutes of all meetings from the date of its founding in 1859 to the present.

There are many, quite possibly the finest, historical anecdotes lying dormant in the memory of descendants of pioneer physicians which no research can uncover without the cooperation of those who know these stories. Events of significance of even 50 years ago are being lost for lack of an effort made to preserve them. Such things as early day epidemics, pioneer surgery, unusual personal experiences, education, travel difficulties, fees, collections, and a thousand other subjects are priceless items in the story of Kansas medicine.

It is hoped that each physician who can recall a story or many events out of medicine's past will contribute them to this Society project and that this will be done soon so the book can be completed by 1959 in time for the centennial celebration. A finished literary effort is not required. The contribution may be a single anecdote, a letter, or a biography. Anything will be appreciated to make this story a complete and an authoritative one.

The second project relating to the history of medicine in Kansas in which the profession is urged to help relates to the Kansas Historical Museum at Topeka. The director is currently constructing a pioneer town with rooms depicting a general store, a home, etc. One such will be a physician's office. This will exhibit authentic medical material, including anything a pioneer physician might have had in his office.

This permanent exhibition is currently being assembled, and a number of rare surgical instruments, saddlebags, and an old examining table have already been contributed. It is hoped that many more items may be made available to give this display the prominence it deserves.

Should any physician be willing to assist with either project, he is urged to write W. M. Mills, M.D., chairman of the Committee on History, Kansas Medical Society, 315 West Fourth Street, Topeka, and describe what he has available so the committee can determine how it might be used. Any cooperation given to either of these projects will be of real service to the Society and much appreciated by the committee.

Opposition to HR 7225

Thousands of words of testimony have been spoken before the Senate Finance Committee in its hearings on HR 7225, the Social Security Amendments Bill passed by the House of Representatives last summer and now under consideration in the Senate.

Many persons from all walks of life have expressed opinions on lowering the retirement age for women from 65 to 62, extending monthly benefits for disabled children beyond the age of 18, extending coverage to self-employed groups, and raising social security taxes. And the most controversial section of the bill, which would make permanently and totally disabled persons eligible to receive benefits at age 50 instead of 65, has been argued at length.

Physicians have been especially critical of this provision, arguing that permanent and total disability is difficult if not impossible to establish, that enactment of this amendment would encourage malingering, that it would be a step toward a program of compulsory federal medical care.

Opposition to the amendment was also expressed, for an entirely different reason, by Mr. Henry Viscardi, president of a manufacturing company known as Abilities, Incorporated, of New York. He believes such consideration for disabled persons is unnecessary, and he should know for he himself was born without limbs.

"I have spent my life close to this problem of disability," he told the committee, "and I have a great faith in solutions which can be obtained in a competitive, free enterprise spirit.

"I was born a crippled child, horribly deformed, with no lower limbs, and I spent the first seven years of my life, consecutive years, in one hospital. . . .

"When I was a child, I remember asking my mother, 'Why me?'

"And she told me that when it was time for another crippled boy to be born into the world, the Lord and his counselors held a meeting to decide where he should be sent, and the Lord said, 'I think that the Viscardis would be a good family for a crippled boy.'"

Possibly a background which includes that type of philosophy gave Mr. Viscardi the impetus he needed to found his company three years ago on an $8,000 loan. In the first year of business the company grew to 59 employees—all disabled persons who could not get work elsewhere. The borrowed capital was repaid, with interest, during the first year, and a profit was realized. During the second year the company grossed more than $400,000 in sales, and during the third year gross sales were in excess of $600,000. The firm produces lace cable assemblies that are a component of the firing mechanism of sabre jets.

Mr. Viscardi now has 169 employees. This fact was of special significance to the Senate committee when he added that every one of this self-supporting group "could qualify for permanent disability under the terms of this law."

These "totally and permanently" disabled persons made a striking contribution to the economy of their community and their country: they produced goods valued at $1,248,700; they received salaries of $668,500; they paid $22,650 in social security taxes, $6,200 in withholding taxes, and $4,830 in disability payments to the state, and they returned $2,067,790 in new wealth to their community. During these three years it would have cost the government $415,850 to have supported these people on relief rolls.

"Their disability is because of prejudice and aversion and ignorance on the part of the industrial and commercial community," Mr. Viscardi testified. "What troubles me is that, should we stigmatize our disabled people and put on an age limit of 50, we might destroy the opportunity for them to be productive, and we might condone some of the ignorance and some of the prejudice that exist which prevent them from exercising their abilities and not their disabilities. . . .

"If we could only, in communities of America and in commerce and industry, shake the ancient superstitions which make us divide our world into able and disabled persons, and the prevailing belief that the man who has lost his limbs is different from other people. From a medical point of view, sure, he is different; but in society and industry it is his abilities that count and not his disabilities. . . .

"The extremes of physical suffering carry a great complement, which is the patience to continue to struggle for the right to be considered the same as the rest of the world, and not different.

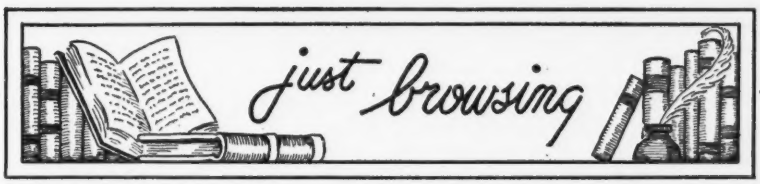
"There is nothing that can be substituted for this basic human right. No honors, no pensions, no parades, no subsidy, can replace the wishes of every person who has known disability to live and work in dignity, in free and open competition with all the world, not as a different person, but rather as the same as others, with varying degrees of weakness and strength and complementary qualities to offset the extremes of physical make-up."

In summing up his opposition to the disability benefits, he added, "I have the belief in other solutions to the problem if we can only try them."

A.M.A. Meeting Next Month

The 105th annual meeting of the American Medical Association will be held in Chicago, June 11-15. Section meeting rooms will be located in University of Illinois facilities, Northwestern University buildings, and nearby hotels. The headquarters hotel will be the Palmer House, and exhibits will be housed at Navy Pier.

The National Medical Civil Defense Conference, sponsored annually by the Council on National Defense, will be held at the Palmer House immediately preceding the A.M.A. meeting, on June 9.



"The great work in which we are engaged demands all our time and all our energies. Let us see to it that we do not allow ourselves to be diverted from the real objects of our pursuit to waste our time and energies in combatting senseless theories, which, left alone, will soonest reach the oblivion to which they are inevitably destined. Let us remember that the aim of the true physician is not the accumulation of wealth, or personal fame or aggrandizement, but the advancement of his science, the perfection of his art, and the emancipation of mankind from the bondage of disease; that he alone is the sole conservator of the public health, and that he alone is charged with the responsible and elevating duty of rescuing his race from the disease and death incurred by their own ignorance and recklessness. This duty and this responsibility is ours.

"We cannot go forth into life without taking upon ourselves all of life's duties and responsibilities. We may ignore or disregard them, but they rest upon us nevertheless, and we shall justly be called to render account for the manner in which we have improved the talents and opportunities which have been intrusted to us. No man can safely allow himself in any pursuit to be governed by self-interest alone.

"If we would not dwarf and cripple the highest and noblest elements of our being, we must seek to be governed by higher and nobler incentives. And what higher incentive to action than the consciousness that its object and its tendency are to prolong human life and rescue it from the thraldom of disease and suffering; to increase its pleasures and diminish its pains; to stay in its course the death-scattering pestilence, and to cause that health and joyousness and thanksgiving shall prevail where otherwise would be death, disease and misery. Can we ask or seek a higher field or nobler work than this?

"Let us study to obtain higher and clearer views of the noble aims of our profession, that we may thereby be led to a more perfect consecration of all our energies to the great and ennobling work in which we are engaged."—*Albert Newman, M.D., President, Kansas Medical Society, 1868.*

Clinicopathological Conference

Severe, Progressive Constipation of the Newborn

CASE PRESENTATION

The case for our consideration today is that of a one-month-old white male who was admitted to KUMC on May 17, 1955, and expired on June 25, 1955.

The parents brought the child in with the comment that he "slept all the time." He had seemed to be normal until five days prior to admission, at which time the parents noticed that he was eating poorly and becoming lethargic. These symptoms became progressively worse during the next five days. His intake of fluid was down to two or three ounces daily, and there was marked oliguria. On the day before admission the child had some seizures characterized by jerking of the head, flexing of the arms, and clenching of the hands. His temperature was 101 degrees. Two small transfusions of blood were given into the gluteal muscles, and subcutaneous fluids were administered at a local hospital prior to his being sent here.

On closer questioning the parents admitted that the infant had been extremely constipated since birth and had been given frequent enemas to produce evacuation of his bowels.

At birth the child had weighed 9 pounds 14 ounces. The remainder of the past medical history is non-contributory.

On admission the patient was a poorly nourished, poorly developed white male who weighed 3925 grams, and who appeared to be acutely and chronically ill. His skin showed extreme pallor, and hematomas were noted in both buttocks. There was a grade II systolic murmur heard over the entire precordium. The abdomen was protuberant and tympanitic, and no bowel sounds were heard. The liver was palpable 5 cm. below the right costal margin.

Numerous pus cells were present in the urine, and there were occasional pus casts. There was no albumin, blood, or sugar in the urine, and urine cultures were all negative. The red cell count was 1,850,000 with 6.1 gm. hemoglobin; the white cell count was 37,400 with 37 per cent polys (29 per cent non-filamented), 6 per cent myelocytes, 8 per cent lymphocytes, and 10 per cent metamyelocytes. There were 83 nucleated red blood cells per 100 white blood cells. The reticulocyte count was 18.8 per cent. The serum non-protein nitrogen was 33 mg. per cent, and the creatinine was 1.3 mg. per cent. The serum electrolytes were: sodium 129 milliequivalents per liter, potassium 5.4 mEq. and chlorides 98 mEq. The red blood cell fragility and Coombs tests were negative. Stool cultures were negative for pathogenic bacteria, but a blood culture on the day of admission was positive for Micrococcus pyogenes var. aureus. On May 24 a blood culture was positive for Escherichia coli. Subsequent blood cultures were negative.

The child underwent surgery on May 18; a greatly distended bladder and peritonitis were found. A cystotomy tube was inserted, and the abdomen was closed. Following surgery the child did poorly for many days, having severe abdominal distention which seemed to be aggravated by oral feeding. Consequently he could not take adequate food by mouth and had to be given intravenous blood, plasma, glucose, and electrolyte solutions. For the first 11 days he received blood almost daily. On May 27 he developed a wound infection at the site of the laparotomy. He had frequent, small, greenish-brown stools almost every day.

For a while the infant gradually seemed to improve and began to take his feedings a little better than previously. His abdominal distention was partially relieved by saline enemas and by colon tube, and he was no longer as lethargic as he had been on admission, albeit he remained somewhat weak.

On several occasions he voided through the penis, and the urinary stream was considered to be adequate. A cystoscopy was done on June 21 and findings were normal, so the cystotomy tube was removed. Subsequent to this he developed a urinary tract infection which yielded a heavy growth of Bacillus proteus. He vomited occasionally while in the hospital, but this was not really a prominent symptom until the last few hours of his life.

During the postoperative period he received several antibiotics, including penicillin, streptomycin, intravenous erythromycin, and intramuscular and oral chloramphenicol. The leukocyte count remained between 20,000 and 30,000 with 10 to 20 per cent of the leukocytes being non-filamented polymorphonuclears. During the last four or five days of life his temperature ranged between 102-103 degrees rectally. Prior to this he had been essentially afebrile.

On June 25 the baby was noted to have extreme abdominal distention, labored respiration, and cyano-

Edited by Jesse D. Rising, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pediatrics, surgery, radiology, and pathology of the University of Kansas Medical Center as well as by the third and fourth year classes of medical students.

sis. It was decided to do a colostomy, but he expired before the procedure could be started. Postmortem electrolytes were normal, but the non-protein nitrogen was 61 mg. per cent.

Dr. Delp (moderator): Dr. Stockdale has tried to anticipate many questions that needed to be asked. Are there any other questions?

R. Leroy Carpenter (fourth year medical student): What about the nature of the bowel movements during the last five days?

Dr. Earl Stockdale (resident pediatrician): They did not change much. He had several small, watery, green bowel movements. Occasionally when the colon tube was inserted he had explosive, large, bowel movements. Rectal examinations were done at frequent intervals, and at no time was a fecal impaction found.

Jay Armstrong (fourth year medical student): Was a blood culture done during the last five days of life?

Dr. Stockdale: No.

Dr. Sloan Wilson (hematologist): Did the blood picture change after the initial count?

Dr. Stockdale: The child was transfused and his hemoglobin went up nicely.

Dr. Wilson: What happened to the reticulocytes and the nucleated red cells?

Dr. Stockdale: On admission there were 83 nucleated red cells per 100 white cells; two weeks later there was only one.

Dr. Wilson: Was a reticulocyte count taken other than the first one?

Dr. Stockdale: No, sir.

Dr. Delp: The white count varied from 37,000 on admission to 56,000 on June 15, which was ten days before death. Then the count gradually dropped to 15,000 on June 24, one day before death.

Nelson Bachus (fourth year medical student): Would you describe the heart murmur, and did it improve upon the administration of blood?

Dr. Delp: It was described as a grade I murmur by one observer and a grade II systolic murmur at the apex by another. Nothing particularly was made of this murmur.

Dr. Antoni Diehl (pediatric cardiologist): Was the fluid in the abdomen cultured at the time of surgery?

Dr. Stockdale: No culture was done at that time.

Dr. Delp: What was done at this procedure?

Dr. Stockdale: A laparotomy was done, and the peritonitis was found. There were adhesions present in the peritoneal cavity, and the bladder was enlarged. Because of this a cystotomy was done.

Dr. Delp: May we see the x-rays?

Willard Brown (fourth year medical student): A flat plate of the abdomen taken on the sixth hospital day shows normal bony structures and a diffuse haziness throughout the abdomen. Considerable gas is present which appears to be in a dilated colon. There is no gas present in the rectal ampula.

The film of a barium enema taken on the seventh hospital day shows the rectum to be of normal caliber; the sigmoid and descending colon are enlarged, and the haustral markings are poor (Figure 1). Again we note a diffuse haziness throughout the abdomen. The lateral view (Figure 2) taken at the same time demonstrates a fusiform swelling which becomes quite marked in the sigmoid area.

On a flat film of the abdomen taken the day of death the liver does not appear enlarged, but there is diffuse haziness throughout the abdomen and marked distention of the colon and intestines with gas. The abdomen appears distended. Again no gas is present in the rectal ampula.

In the chest film the lung fields appear clear, and there is no inflammatory process apparent. The heart is normal in size and configuration for a patient of this age.

Dr. Delp: Dr. Youngstrom, do you have any comment concerning these films?

Dr. Karl Youngstrom (radiologist): No, I think that they have been well described. I might add that the fluoroscopy showed inertia in emptying the bowel.

Dr. Delp: Mr. Carpenter, we want your summary and differential diagnosis.

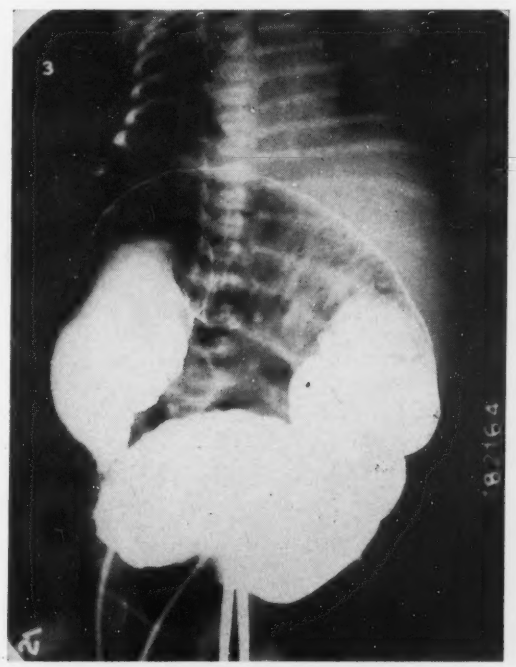


Figure 1

DIFFERENTIAL DIAGNOSIS

Leroy Carpenter (fourth year medical student): The patient for consideration today was a one-month old male who had had constipation requiring frequent enemas since birth. Otherwise he was apparently well until five days before admission, when he began to take feedings poorly, became lethargic, oliguric, febrile, and had generalized seizures. He entered KUMC acutely and chronically ill, weighing one pound less than his birth weight. His abdomen was protuberant and tympanitic. There was absence of bowel sounds and a faint heart murmur. Laboratory studies revealed pyuria, severe anemia, reticulocytosis and a positive blood culture for hemolytic staphylococci.

A laparotomy was performed on the day after admission. The protocol mentions that distention of the bladder and peritonitis were found, and that a cystotomy tube was inserted. The patient received blood, fluid, and intensive antibiotic therapy following surgery. He did poorly for a few days, then began to improve. He voided well, and cystoscopy was normal. Five days before death he again developed a temperature of 102 to 103 degrees, marked abdominal distention, and labored respirations. He expired prior to a planned colostomy five weeks after admission.

I shall base my differential diagnosis on the causes of chronic progressive constipation of the newborn. Occasional causes of constipation of this type are spinal cord lesions such as cord injuries, meningomyeloceles, and spinal cord tumors. I rule these out because of the lack of any history of spinal cord injury and the lack of x-ray or physical findings.

This leaves us with the consideration of megacolon. Functional megacolon never occurs in this age group. Organic megacolon caused by lesions occurring in the lower colon and rectum, such as hemangiomas, primary sarcomas, carcinomas, polyps, congenital bands and valves, or anal sphincter abnormalities can be ruled out on the basis of the rectal examinations and x-ray findings.

The third type of megacolon is that called Hirschsprung's disease or aganglionic megacolon. It occurs in one out of every 20,000 to 30,000 live births and is ten times more frequent in males than in females. The outstanding features of this disease are progressive constipation starting at birth, anorexia, weight loss, anemia, bladder distention, and occasionally megaloureter. Ninety per cent of such cases have narrowing of the recto-sigmoid junction. Temporary relief is given these children by enemas, but this is never permanent. I believe that this patient had typical Hirschsprung's disease. He had chonic progressive constipation since birth. The fecal retention may have resulted in pressure necrosis and perhaps perforation of the intestine with resulting peritonitis and septicemia. The terminal event, I feel, was a combination of acute colitis, recurrence of the peritonitis, and septicemia which led to his death.

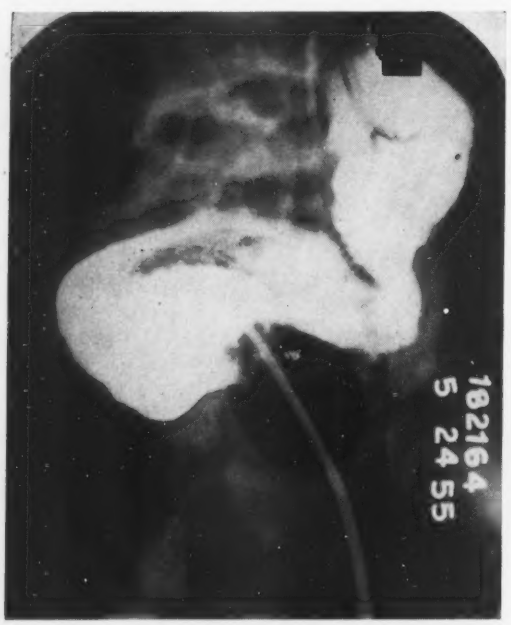


Figure 2

CLINICAL DISCUSSION

Dr. Delp: Mr. Bachus, why was this patient lethargic? Apparently he was lethargic from the day he got home from the hospital, about the sixth or seventh day of life, and had to be awakened for his feedings. Lethargy was prominent when the child arrived here.

Mr. Bachus: I thought originally that his lethargy was due to his septicemia, and was increased by his anemia and dehydration. But if he was lethargic from the time he came home from the hospital that would not explain it. I have no other explanation.

Dr. Delp: If his lethargy had started ten days before he came in, it could be explained by his sepsis?

Mr. Bachus: Yes, sir.

Dr. Delp: Mr. Belzer, what do you think about the problem?

David Belzer (fourth year medical student): I agree with Mr. Bachus. It might be caused by the septicemia that this child had, and aggravated by the dehydration on the basis of decreased intake of fluid.

Dr. Delp: Mr. Bare?

Chester Bare (fourth year medical student): I agree with what has been said, but the anemia could cause it also.

Dr. Delp: Mr. Carpenter?

Mr. Carpenter: He had oliguria and I would add, along with the other causes, the possibility of retention of nitrogenous wastes.

Dr. Delp: His nonprotein nitrogen was 33 when he came in. Mr. Brown, can you think of something else that might make the patient lethargic?

Mr. Brown: It could be his CO_2.

Dr. Delp: His first CO_2 was 24.4 mEq; the next one was down to 23.2, and the one done on the 25th was 7.2. Anything else, Mr. Armstrong?

Jay Armstrong: I was thinking about potassium, but it is normal here.

Dr. Delp: Well, he had several potassium determinations; the first one was 5.4 mEq/L and the next one was 4.9. The last blood specimen was hemolized. Mr. Alcox, why did this patient have seizures?

Leroy Alcox (fourth year medical student): Seizures are frequent in young children with high fevers.

Dr. Delp: Are you perfectly satisfied with that explanation? Can't you be a bit more exact?

Mr. Alcox: On the basis of the laboratory findings there is nothing to explain it.

Dr. Delp: Mr. Bachus?

Mr. Bachus: I thought this was a febrile convulsion.

Dr. Delp: Well, of course we don't know whether he had fever with the first one, but he apparently had fever when he first got to the hospital. Mr. Alcox, how can we explain the fact that this patient had peritonitis?

Mr. Alcox: Peritonitis is frequent in children with Hirschsprung's disease. Enemas or other attempts to lavage the intestines easily perforate it.

Dr. Delp: Do you have any other ideas, Mr. Bachus?

Mr. Bachus: I don't think the patient perforated. I think that the bacteria were transmitted through the bowel secondary to dilatation.

Dr. Delp: By osmosis?

Mr. Bachus: Well, perhaps a pressure necrosis would let them through the wall.

Dr. Delp: I see, and the wall became more permeable under those circumstances. Mr. Belzer, do you accept that?

Mr. Belzer: Well, it is a possibility, but I also think the septicemia could explain it.

Dr. Delp: What was the origin of the septicemia?

Mr. Carpenter: I think this lad had colitis. He had an infection in his bowel, and the bacteria got into the peritoneum that way. He had large fecal masses, distention, and irritation of the bowels with colitis and ulcerations.

Dr. Delp: Mr. Bare, what do you think that antibiotics had to do with this patient's signs and symptoms?

Mr. Bare: They undoubtedly account for the negative blood and urine cultures that were found during his hospitalization.

Dr. Delp: Anything else?

Mr. Bare: The chloramphenicol could account for some of the shift to the left in the blood count.

Dr. Delp: Mr. Brown, what do you think?

Mr. Brown: There are several cases reported in which antibiotics were the cause of the colitis.

Dr. Delp: Mr. Carpenter?

Mr. Carpenter: Antibiotic colitis is a strong possibility with change in flora to resistant forms.

Dr. Delp: Do you want to change your diagnosis now?

Mr. Carpenter: I will stick to Hirschsprung's disease with a secondary colitis.

Dr. Delp: Mr. Alcox, why did this child have a distended bladder?

Mr. Alcox: In 50 per cent of all cases of Hirschsprung's disease, there is an associated dilatation of the bladder.

Dr. Delp: You heard that there was a cystoscopic examination here and that the lower urinary tract was normal. What about the distended bladder, Mr. Bachus?

Mr. Bachus: I was accounting for it in much the same way. Certainly he didn't have any lower urinary tract pathology to produce the distended bladder, and one might think of a primary bladder hypotonia.

Dr. Delp: Mr. Carpenter?

Mr. Carpenter: I was going to postulate that the peritonitis caused this bladder distention.

Dr. Delp: Mr. Armstrong, why was this patient dyspneic?

Mr. Armstrong: Probably on the basis of the abdominal distention to start with. The lung fields look pretty good.

Dr. Delp: Mr. Brown, what caused the dyspnea?

Mr. Brown: Didn't you say that his CO_2 was 7?

Dr. Delp: I did!

Mr. Brown: Probably acidosis.

Dr. Delp: Mr. Bachus?

Mr. Bachus: His anemia could also be another factor.

Dr. Delp: When he was extremely dyspneic his hemoglobin was 50 per cent. Was he dyspneic all the time, Dr. Stockdale, or just the last two or three days?

Dr. Stockdale: Severe dyspnea was present only in the last few days.

Dr. Delp: Have you any other ideas, Mr. Bare?

Mr. Bare: I don't have any.

Dr. Delp: Dr. Miller, I would like to have your comments concerning this patient.

Dr. Herbert Miller (pediatrician): I would like to

TRANS WORLD AIRLINES

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respiratory infections

January and his associates[1] have written on the use of tetracycline (ACHROMYCIN) to treat 118 patients having various infections, most of them respiratory, including acute pharyngitis and tonsillitis, otitis media, sinusitis, acute and chronic bronchitis, asthmatic bronchitis, bronchiectasis, bronchial pneumonia, and lobar pneumonia. Response was judged good or satisfactory in more than 84% of the total cases.

Each month there are more and more reports like this in the literature, documenting the great worth and versatility of ACHROMYCIN. This antibiotic is unsurpassed in range of effectiveness. It provides rapid penetration, prompt control. Side effects, if any, are usually negligible.

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[1]January, H. L. et al: Clinical experience with tetracycline. *Antibiotics Annual* 1954-55, p. 625.

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ask what kind of enemas were given to the baby at home?

Dr. Delp: We don't know.

Dr. Miller: If these were tap water enemas, this could possibly account for the low sodium that this baby had. He did have an acidosis according to studies that were done on admission, with the base totaling up to 134 milliequivalents and the total anions in the neighborhood of 144 milliequivalents. This would suggest that the baby had some respiratory acidosis when he came in. He was distended and perhaps did have some trouble blowing off carbon dioxide as a result of the marked abdominal distention. The low sodium might have been accounted for by repeated enemas which would tend to draw sodium out of the body if tap water were used.

Figure 3. A shaggy pseudomembrane covers the mucosa of the hypertrophied and dilated descending and sigmoid colon. Arrow indicates the beginning of the rectum where the lumen begins to narrow to normal caliber and the pseudomembrane disappears. A small band of anal skin is included on the lower end of the specimen (reduced ¼).

Dr. Delp: On the second day, Dr. Miller, the sodium was 126 and the chloride was 94.

Dr. Miller: I would think that these are probably correct. In my opinion the baby's sleeping all the time was related to the infection. What caused the convulsions I do not know, unless they resulted from fever. It is easy for babies to have convulsions when they have considerable fever. I cannot explain why this is so.

I have no reasons to disagree with Mr. Carpenter with regard to what was going on in this baby. I think that he is very likely right.

Dr. Delp: Dr. Allbritten, do you have any comments about this diagnosis? Do you disagree with Mr. Carpenter?

Dr. Frank Allbritten (surgery): No, I agree with the diagnosis. I think that it was not clear at the time of exploration as to the extent of the baby's adhesions and peritonitis. I assume it was generalized peritonitis. There was no comment about the colon's having been identified at the time of operation, and it is conceivable that there was a mass of adhesions and fibrinous material so that one could not delineate the small and large bowel, although the bladder could be seen as an anatomical structure.

Dr. Delp: One of the medical students drew a picture in this chart indicating a ridge-like mass that extended from the upper left quadrant to about the middle of the right flank. Do you think this would mean anything so far as the physical findings are concerned?

Dr. Allbritten: Yes, it would be suggestive of a tremendously distended transverse colon.

Dr. Delp: Now may we have the pathologist's report?

PATHOLOGY REPORT

Dr. Harlan Firminger (pathologist): At the time of autopsy this child was about normal in height, measuring 23 inches. He was distinctly undernourished, weighing only 4300 grams, and his abdomen was greatly distended. There was a healed scar over the lower abdomen and a healing cystotomy wound. When the abdomen was opened the bowel loops were clearly adherent, and everything was bound together in a mass by fibrinous adhesions. In addition there was an abscess 4 cm. in diameter in the pelvic region.

The major item of interest was the colon. It was a tremendously dilated structure occupying much of the abdomen. It had thick walls and was distinctly hypertrophied as well as dilated. The lumen of the colon narrowed gradually just above the rectum to about normal caliber (Figure 3).

The mucosa of the colon was covered with a white pseudomembrane which, when scraped off, revealed a rather granular underlying surface. The pseudo-

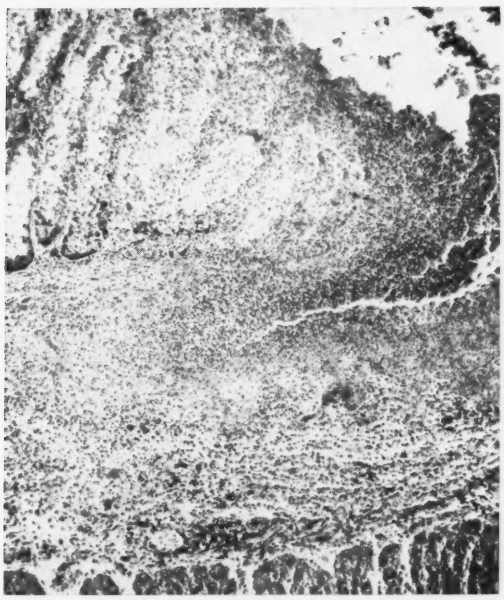

Figure 4. Pseudomembranous colitis with edge of an ulcer extending into the submucosa. There is necrosis particularly of the superficial mucosa which is covered by a fibrinopurulent membrane. At the right margin the marked inflammatory process extends through the mucosa into the submucosa. The underlying muscularis propria, incompletely shown, is much thickened (hematoxylin and eosin stain).

membrane covering the colonic mucosa disappeared in the rectum.

A microscopic section (Figure 4) shows the pseudomembranous covering of the colon with many polymorphonuclear leukocytes and inflammatory cells throughout all layers, some destruction in the upper layers of the mucosa, and considerable edema and inflammatory infiltration of the submucosa. In one area there is an ulceration of the colon; there were a number of such ulcerations. In three places we found actual perforations of the colon extending out into the adjacent peritoneal area.

The volume of the urinary bladder was only moderately increased, but the wall of the urinary bladder was three or four times normal thickness. A section of the wall of the bladder showed the thickening to be the result of hypertrophied muscularis and submucosa.

There were several lesions in the lungs. One was an organizing embolus in the pulmonary artery at the hilus of the left lung. We also found a cystic area of old infarction at the base of the right lung which was adherent to the diaphragm. There were scattered yellow areas which we interpreted as aspiration pneumonia; at the time of autopsy there was vomitus in the trachea and in the mouth.

The liver was slightly enlarged, weighing 300 grams. It contained only a small amount of fat. There were pigmented macrophages and Kupffer cells containing a golden brown hemosiderin pigment everywhere. This indicated hemosiderosis. There were also foci of hematopoesis, and the bone marrow was hyperplastic.

There was a marked involution of the thymus. It weighed only 1.5 grams.

The heart was enlarged, weighing 38 grams, which is almost twice the normal weight for this age.

Regarding the Hirschsprung type of megacolon, there is ordinarily a constricted, aganglionic segment of bowel at the region of the rectosigmoid, and indeed that was the situation in this case (Figure 5, top). Above this level, in the hypertrophied portion of the megacolon, Auerbach's plexus is intact (Figure 5, bottom).

As has been suggested, this is sometimes associated with distention of the urinary bladder. Both the urinary bladder and the colon are supplied by the pelvic parasympathetic plexus, and it seems likely that deficiency of these nerves would involve the bladder as well as the colon. At times this has been found.[3] In this case we have been unable to confirm this despite many sections. We could see nothing in the bladder and urethra to explain the distention, and we could

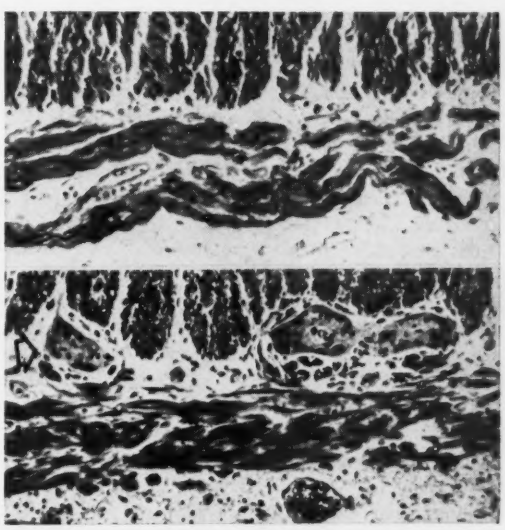

Figure 5. Note the absence of Auerbach's plexus in the aganglionic segment of the rectum (top) in contrast to the large ganglia, indicated by arrow, in a section from the hypertrophied and dilated colon (bottom) (hematoxylin and eosin stain).

not say that it was necessarily comparable to the change in the colon.

The pseudomembranous colitis is similar to the two cases which Moriarty and Ramsey[1] reported in babies with megacolon who had been given large doses of antibiotics. Presumably this was a matter of changing flora and stasis of the material in this area.

The enlarged heart is difficult to explain. Dr. Miller has already referred to the possibility that this baby was given tap water enemas at home. In addition to losing sodium, there may have been absorption of water which could produce hydremia. Another factor may have been the anemia, but this heart was hypertrophied out of all proportion to what is normal in a child of this age. With respect to the anemia, there was hyperplasia of the marrow with abundant red cell formation. Most of these children do have anemia, though I am not quite sure what the etiology is. I doubt that it is suppression of the marrow. The hemosiderosis is probably attributable to the transfusions which this child had received.

Dr. Delp: There have been allusions to the fact we now have for this syndrome an explanation which did not exist a few years ago. I am old enough to recall vividly and bitterly the number of such patients that I encountered when I was an intern and resident in which the outcome was invariably just as unsatisfactory as in this case.

I would like to clear up a few things in the time remaining by calling on Dr. Allbritten, Dr. Miller, and Dr. Valk.

Dr. Allbritten: There has been considerable change in the treatment of this disease in the past few years. Previously the treatment was directed toward the dilated segment of the colon, and surgical attempts were made to resect large areas of the dilated portion of the colon and do end to end anastomosis. To everyone's disappointment, the dilatation promptly recurred. The attempts were directed toward the obvious area rather than to the area of functional obstruction. In 1948 Whitehouse and Kernohan,[5] and later Swenson with his coworkers,[4] approached the disease from a different standpoint and started the direct surgical attack on the obstructed area. They resected the aganglionic segment of the bowel and anastomosed the colon to the anal area. This procedure has proved to be more satisfactory.

One thing that has been commented on, and which needs repetition, is that the area that is dilated was examined for abnormalities, but no abnormality was found. Regarding this patient's bladder, I wonder if we were not looking in the wrong area in the bladder, and should have been looking at the site of obstruction rather than at the site of dilatation.

Dr. Firminger: We did examine the urethra, and ganglia were present down into the prostatic urethra at least.

Dr. Allbritten: Even through the membranous urethra and sphincter?

Dr. Firminger: Our microscopic sections include the sphincter and part of the prostatic urethra. The remainder of the urethra was grossly normal.

Dr. Delp: Dr. Valk, do you have any comments?

Dr. William Valk (urologist): When one encounters distention of the bladder and pus in the urine, the normal procedure is to pass a catheter into the bladder to relieve this distention. This type of bladder can obstruct the recto-sigmoid and cause severe dilatation of the colon. It can blot out the venous return in the pelvic veins and cause edema of the extremities. I am sure that this child had primary Hirschsprung's disease, but I am also sure that he might have been greatly improved by simple passage of a catheter before surgery. This would have relieved his distention and distress a great deal. As has been said, urinary tract anomalies are associated with Hirschsprung's disease, but it is a little different from the bowel and gastrointestinal tract as far as innervation is concerned. One can totally denervate the ureters and the bladder without producing a picture similar to Hirschsprung's disease. A denervated ureter does not distend and become a greatly dilated structure as the colon does. I believe that it is probably a little different mechanism than aganglionic disease of the colon.

Dr. Delp: Dr. Miller?

Dr. Miller: The English have recently shown that, in ten babies who had Hirschsprung's disease, the colon is not large and dilated in the first two or three months of life. Might not the bladder have been enlarged because of peritonitis instead of Hirschsprung's disease?

Dr. Valk: At operation the bladder was a thin, friable, dilated structure. This is extremely unusual in the absence of organic or neurogenic obstruction of the bladder. Hypertrophy of the bladder indicates that oliguria must have been present at birth or even from the seventh month of intrauterine life when the fetus first starts to pass urine into the amniotic fluid.

Dr. Miller: You think the bladder was involved even though the ganglion cells appear in normal amount?

Dr. Valk: I don't know.

Dr. Delp: Any final comments or questions?

Dr. Youngstrom: Swenson and Fisher[2] have recently reported on dilated ureters associated with this condition, and they theorize that the lesion might be in the cord rather than in the bladder or urethra. So there seems to be room for further thought on the etiology of this condition.

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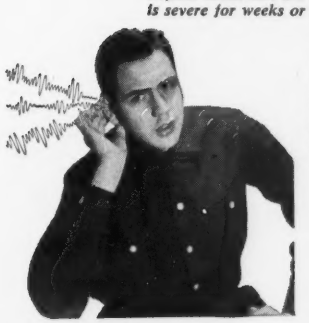
III. Ménière's Syndrome

1. Paroxysmal Whirling Vertigo. *This consists of sudden attacks of dizziness, often when the patient is at rest or asleep. The patient may feel that he himself is whirling or that fixed objects about him are whirling. The attack usually lasts for a few minutes; occasionally it is severe for weeks or subacute for months.*



2. Subtotal Hearing Loss. *Deafness will usually affect the high tones and it may be unilateral or bilateral. Sometimes the hearing loss is severe and also progressive.*

3. Tinnitus. *This is usually unilateral and present in the ear with greater hearing loss and is without a definite pattern.*

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1. Severe paroxysmal vertigo which may be of two types; either the patient feels that he is whirling or that objects about him are whirling.
2. Fluctuating subtotal hearing loss, usually affecting the higher tones, is noted at the same time as vertigo.
3. Tinnitus, usually unilateral, is associated with the deafness and dizziness.

With Ménière's syndrome there is no definite localization[2] by the Bárány (vestibular reaction) test and results of the caloric test are not diagnostic. Physical examination should rule out disease of the central nervous or cardiovascular systems before a diagnosis is made.

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1. DeWeese, D. D.: Symposium: Medical Management of Dizziness. The Importance of Accurate Diagnosis, Tr. Am. Acad. Ophth. *58*:694 (Sept.-Oct.) 1954.
2. Jackson, C., and Jackson, C. L. (editors): Diseases of the Nose, Throat, and Ear, Philadelphia, W. B. Saunders Company, 1945, pp. 368; 414.
3. Queries and Minor Notes: Ménière's Syndrome, J.A.M.A., *141*:500 (Oct. 15) 1949.

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Dr. Delp: Hirschsprung's disease should occupy high priority in a differential diagnosis of progressive constipation in the newborn. Such a statement becomes even more reasonable today with newer knowledge concerning the true character of the malady and the gratifying results from prompt surgical treatment. Only a moment's thought will suggest, as in this case, the danger of salt depletion and water intoxication which may result from retained water enemas in such a patient. The recently recognized relationship of megacolon and megaloureters is given further legitimacy by this case.

PATHOLOGICAL ANATOMICAL DIAGNOSIS

Primary

Congenital megacolon with hypertrophy and dilatation of the sigmoid, transverse and ascending colon with multiple ulcers, advanced.

Multiple ulcers of the ileum and jejunum, slight (history of abdominal distention and constipation for six weeks before death).

Hypertrophy of the urinary bladder (history suggestive of megalobladder).

Healing midline incision of anterior abdominal wall; healing cystostomy wound and sinus tract (history of distention of the urinary bladder and pelvic peritonitis with performance of cystostomy).

Organized emboli in smaller branches of the pulmonary artery with old cystic infarct of the lower lobe of the right lung and overlying fibrous pleural adhesions.

Loculated small abscess in pelvis (history of incision and drainage of wound abscess four weeks before death).

Malnutrition, height 23 inches, weight 9½ pounds.

Diffuse pseudomembranous colitis with penetrating ulcers and local acute fibrinopurulent peritonitis (history of treatment with several antibiotics including aureomycin).

Diffuse serofibrinous peritonitis with focal areas of angulation of the small bowel and penetrating ulcer of the colon.

Fatty metamorphosis of the liver, 300 gms.

Involution of the thymus, 1.5 gms.

Hyperplasia of the bone marrow of the sternum and vertebrae.

Foci of extramedullary hematopoiesis in the liver and spleen, slight.

Hypertrophy of the heart, 38 gms.

Hemosiderosis of the liver and spleen, slight (history of anemia and repeated small blood transfusions).

Dilatation of acini of pancreas, slight (history of dehydration).

Recent puncture wounds and ecchymoses of both femoral regions and of the scalp and healing incisions over both medial malleoli (history of repeated venipunctures).

Gastric contents in the oropharynx and tracheobronchial tree.

Foci of aspiration pneumonia.

Partial atelectasis of the upper and lower lobes of both lungs.

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3. Swenson, O.; MacMahon, H. E.; Jaques, W. E., and Campbell, J. E.: A new concept of the etiology of megaloureters, New Eng. J. Med. 246:41-46, 1952.

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Pamphlet on Quacks

To help the public identify some of the devices, gadgets, and machines used for so-called "treatments" or "cures" of many diseases, the A.M.A.'s Bureau of Investigation has issued a new pamphlet on mechanical quackery. A check list for identification of quacks in the local community is included.

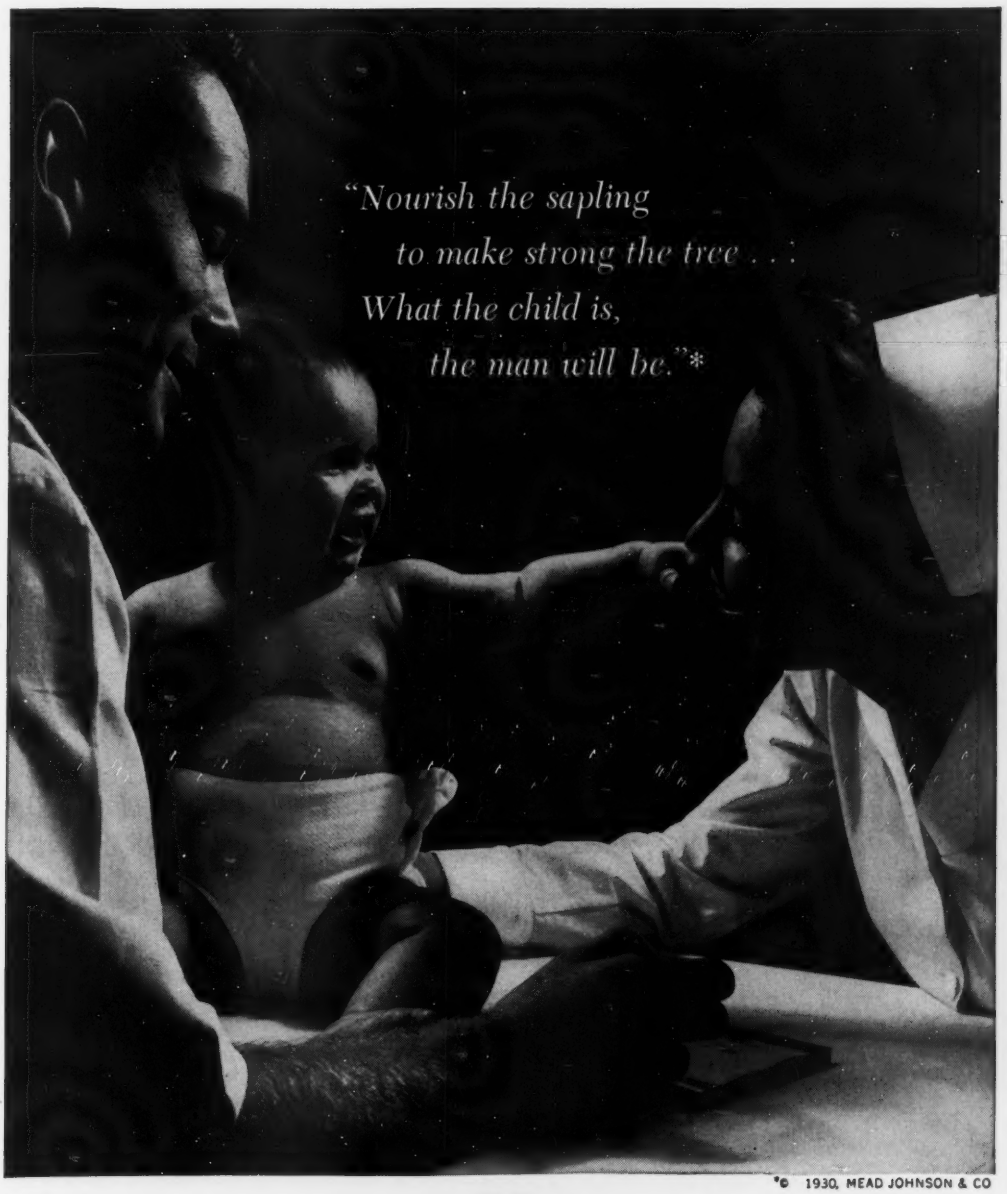
The pamphlet will be distributed primarily at A.M.A. exhibits on quackery at medical society meetings, health fairs, museums, etc. It will also be sent by mail to those who request copies from the bureau.

Information on Adoptions

Proceedings of a conference on adoption held last year by the Department of Health, Education, and Welfare are published in a report, "Protecting Children in America," recently released by the Children's Bureau. Attended by representatives of the medical, legal, and social professions, the conference explored ways to eliminate the "black market" in babies.

Copies of the pamphlet may be secured from the Children's Bureau, Washington 25, D.C.

The Series H Savings bond, issued only since mid-1952, will pass the three billion dollar mark in sales before it is four years old.



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PHYSICIANS' ACTIVITIES

Dr. Jacob T. Whallon, Wichita, who recently took up art as a hobby, painted an oil picture which was chosen for exhibit at the third annual art show in Wichita recently.

Dr. R. Dale Dickson, Topeka, was a member of the faculty at a postgraduate course sponsored by the American College of Allergists in New York in April.

Dr. Eldon S. Rich, Buhler, has announced plans to close his office there to go to Newton on June 1 to join the staff of the Bethel Clinic. He will practice obstetrics and general medicine.

Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, is a member of a team of six physicians now conducting a series of meetings in Japan for medical schools and medical societies. The educational program is sponsored by Unitarian Service Committee, Inc., and will last for six weeks.

Dr. J. S. Menaker, Wichita, presented a paper, "Psychosomatic Gynecology," at a meeting of the American Academy of Obstetrics and Gynecology in Memphis in March.

Three Kansas City physicians, **Dr. A. J. Rettenmaier, Dr. C. J. Mullen,** and **Dr. W. J. Feehan,** have been named to an advisory and planning committee for Creighton University, Omaha.

Dr. Enos A. Nelson, Phillipsburg, was honored recently by the Masonic order in that city on the completion of 50 years of membership.

Dr. Alvin Y. Wells, Winfield, was speaker at a recent meeting of the Cowley County Medical Assistants' Society. He discussed serums and vaccines.

Dr. George M. Gray, Kansas City, who was honored recently on the occasion of his 100th birthday, was the subject of a number of feature stories, especially in Kansas City papers. In one photograph Dr. Gray was pictured while reading the JOURNAL OF THE KANSAS MEDICAL SOCIETY.

Dr. Robert M. Brian, El Dorado, spoke on "Radioactivity and Isotopes" at a recent meeting of the El Dorado Hospital Auxiliary.

Health officers for a number of counties were named recently by the respective boards of county commissioners. Those announced are: **Dr. L. G.**

DEATH NOTICES

ROBERT L. VON TREBRA, M.D.

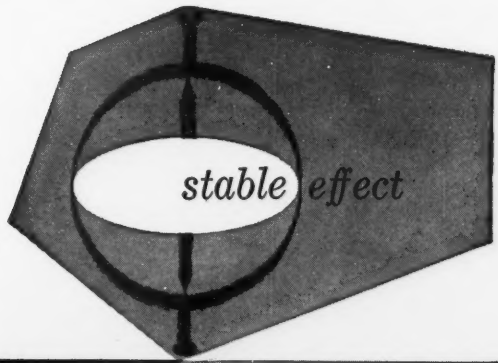
Dr. R. L. Von Trebra, 86, a retired Chetopa physician, died in a Parsons hospital on March 11 after having been a patient for several months. He was graduated from Keokuk Medical College in Iowa in 1897 and practiced there for a while, moving to Kansas in 1901. He was associated in practice with his sister, Dr. Ernestine Von Trebra. He was interested in Society activities, holding membership in the Labette County Medical Society, and also had a number of civic interests. He had served as a councilman and as mayor of Chetopa and had also been elected to the state legislature.

JOHN ARTHUR HIBBLER, JR., M.D.

Dr. J. A. Hibbler, Jr., 44, a member of the Wyandotte County Medical Society, died at Wheatley Provident Hospital, Kansas City, on March 12 after suffering a cerebral hemorrhage several days earlier. He was a graduate of Meharry Medical School, class of 1935, was a resident surgeon at General Hospital No. 2, Kansas City, Missouri, for three years, and went into private practice in 1939.

MAURICE S. WESSELL, M.D.

Dr. M. S. Wessell, 54, who formerly practiced in Hiawatha and had been in practice in Burlington since 1954, suffered a heart attack and died on March 25 while in Hot Springs, Arkansas. A graduate of Albany Medical College in 1927, Dr. Wessell was interested in military medicine and served as a flight surgeon from 1940 to 1947. From then until 1951 he was an associate professor of surgery at the University of Arkansas School of Medicine, Little Rock, and chief surgeon at the Veterans Administration Hospital there. He was a member of the Coffey County Medical Society.



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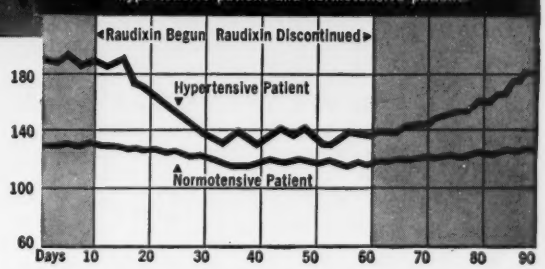

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Dr. Adelbert R. Chambers, Iola, has announced that he is a candidate for the Republican nomination to the office of Allen County representative in the state legislature.

Dr. Peter K. Wiens, who graduated from the University of Kansas School of Medicine last year and has completed internship at St. Luke's Hospital, Kansas City, Missouri, has begun practice in Minneola.

Dr. Clyde B. Trees, president of the Shawnee County Medical Society, has accepted appointment to the Topeka Board of Education's 20-member advisory committee.

Dr. George W. Davis, Ottawa, recently observed the 60th anniversary of his graduation from the University Medical College of Kansas City, Missouri.

Dr. Wayne O. Wallace, Atchison, was speaker at a dinner meeting for the public sponsored by the Atchison County Chapter of the American Heart Association. He also showed a film, "The Valiant Heart."

Dr. Charles S. Huffman, Columbus, was honored by the Masonic Lodge in his home community recently and was presented with a 50-year membership pin.

A feature story in the *Coldwater Western Star* recently paid tribute to **Dr. R. A. J. Shelley** who has been practicing since 1903, first at Waldron, then at Protection and Zenda, and in Coldwater for the past 32 years.

Dr. Val Converse, formerly of Kansas City, moved to Horton on March 1 and is now associated with the Horton Hospital and Clinic.

The Sharon Springs community announces that its seven-year search for a physician ended recently when **Dr. A. R. Cuadrado,** who had been practicing in Colby, moved there to practice.

Dr. Melvin Masterson, who practiced in Richmond, Missouri, before entering service with the Air Force in 1954, was discharged recently as flight surgeon at the Air Force Base at Waco, Texas, and has opened an office in Louisburg.

The Sedgwick County Chapter of the American Academy of General Practice announces the election of the following officers: president-elect, **Dr. Bruce P. Meeker;** vice-president, **Dr. Jack G. Phipps;** secretary-treasurer, **Dr. Harry T. Hidaka,** and member of the board of directors, **Dr. Harold L. Low.**

Dr. William L. Valk, of the University of Kansas Medical Center, was one of the speakers at a meeting of the Urologic Society of Mexico at Mexico City last month.

The Samuel J. Crumbine medal, bestowed by the Kansas Public Health Association for outstanding service to public health, was awarded to **Dr. Ralph I. Canuteson,** Lawrence, last month.

Dr. Garland O. Wellman, on leave of absence from the Sedgwick County Society and now in Texas City, Texas, was elected to fellowship in the American College of Surgeons recently and also became a diplomate of the American Board of Surgery.

An invitation to participate in the President's Conference on Occupational Safety, to be held in Washington, D. C., May 14-16, has been accepted by **Dr. Leslie L. Saylor,** Topeka.

Dr. Lennert B. Mellott, who has served in the medical department of the Sunflower plant for several years, has announced plans to return to semi-private practice in Bonner Springs. He will continue to serve at the plant three mornings a week.

Dr. Karl Stock, Topeka, returned recently from Baltimore where he attended a meeting of Wilmer Eye Residents at Johns Hopkins University.

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"If I wished to show a student the difficulties of getting at truth from clinical experience, I would give him the history of epilepsy to read."

Oliver Wendell Holmes

There are many wonderful new drugs coming out every month or so for the control of epilepsy. Each is greeted with great acclaim, then after a time it, too, takes its true place in the treatment of epilepsy. It is interesting that while each of these new drugs is heralded as being the one which will free the poor sufferers from their scourge, none of them, when viewed in their true perspective, seem to do so. A review of the history of the treatments of epilepsy and the drugs and treatments that in prior times were to have cured man of epilepsy would be interesting and would give added impetus to the words of Oliver Wendell Holmes.

In this paper treatment will be the main subject of discussion; theories as to etiology will be mentioned only insofar as they pertain to the rationale, if any, of the therapy in question.

Judging from early historic writings and pre-historic findings, the old saying should be changed to read, "Taxes, the poor, and epilepsy we have always with us." During the period of time from the neolithic to the present about everything animal, vegetable, or mineral has been used in the treatment of epilepsy. Depending upon the theory under which a patient was treated, material was poured into a patient or drained out; this not being possible, it was rubbed into or hung upon a patient. Incantations for and against the patient were used, and many a poor epileptic has lost parts of his anatomy in attempts at treatment. At one time or another, the conduct of the poor epileptic was controlled more rigidly than that of an eager virgin.

For a general outline of the subject of folklore history of epileptic treatment, five general categories seem possible: (1) the general way of life by which an epileptic must abide; (2) spiritual treatments, including devils and gods; (3) animal treatments, including man and lower forms; (4) vegetable treatments, including all drugs grown on the earth; (5) mineral treatments, both in their pure and adulterated forms.

It must be understood that three basic principles were used in treatment, i.e., magic or superstition, rational explanation, and empiricism.[1] To differentiate between these is difficult; since this is not written at the time of the prescribers, no distinction can be made.

As a prerequisite to living without seizures an epileptic was told to stay away from swamps, rivers, or any moist area.[2] He was told to remain away from too much wind, sudden changes in temperature, or loud noises, for all these things would bring on the disease.[3] He was to sleep, but not too soundly or during the day. Exercise, of course, was good, but only in moderation, taken just as prescribed and when scheduled.[4] As Galen says, "Speaking generally, I recommend abstinence from daily or immoderate use of such food as engenders unhealthy humors or as causes constipation or flatulence, and is hard to digest."[5] Galen is safe in such generalities. More specifics are listed later pertaining to foods.

These above prescriptions were good because epilepsy was said to be caused by various humors. If the brain were too humid, humors rising to the brain would make the brain too moist, or the heat would make the humor too thin, or cold would make the phlegm too thick. These were certain things that would cause bile to congeal in the brain. Exercise would keep the humors flowing.[1, 4, 6, 7]

Some said that baths should be abhorred because of moisture,[5] whereas others thought them beneficial for their purifying effects.[8] Efforts at dehydrating the patient and at fasting were made; however, no explanation or reason other than spiritual or humoral was given until the 20th century.[2, 8, 9, 10]

It is interesting to note that diet has had much attention throughout the ages. The Arabs anticipated the ketogenic diet for whatever reason, in that long ago they prescribed a diet of fat meats "to the point of disgust and nausea."[6] During the middle ages much talk, but no progress, was made regarding diet. Gowers himself includes a discourse on the subject. It was not until the 1920's that the reason for a ketogenic diet was given.[9]

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Dougherty is now serving his internship at General Hospital, Kansas City, Missouri.

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The use of wine or alcohol in any form was controlled. There were those who advised small quantities;[1] however, none encouraged its use.[3, 10, 11] This is one point upon which it seems that even the superstitious agree with the physicians of the day, who definitely concur with modern day therapy.[9]

Whether or not an epileptic should marry is a question that has been discussed for ages. Granted, views as to why one should or should not marry differ from learned discourses on eugenics to the individual effects of intercourse on the course of epilepsy, but each author has had strong feelings on the subject throughout the ages.[1, 2, 10]

Gowers in 1885 states, "In the interest of the individual, of the family, and of the race, it is therefore desirable to discourage as far as possible the marriage of epileptics."[10] Hector Boece, writing about 1550, indicates that the Scottish clans thought more strongly about the matter. He recommended that a male epileptic be "instantly gelded and a woman kept from all company with men and if she were found to be with child, she and her brood were buried alive."[14]

The sexual act itself was mentioned often, and it was said to be closely allied with epilepsy. Some said that coitus cured epilepsy,[5] others said it caused epilepsy.[8, 15] Temkin quotes the Hippocratic papers: "Coitus is a slight epileptic attack."[4] Kanner quotes, "Coitus brevis epilepsia est."[6]

Castration was practiced. At some periods it would seem to have been used for the purposes of a cure,[15] at others as a form of eugenic hygiene,[10] and still again as a punishment.[14] It is rather difficult to tell from the literature just which was the reason. Oophorectomy for a cure was advised as late as 1880. There is also "the strange unverified and not quite probable belief that Napoleon Bonaparte had an epileptic attack after each intercourse." Kanner tells of a certain Turk, Sultan Murad III, who owed his epilepsy to over indulgence in the harem.[6]

Pliny believed that intercourse was good for epilepsy.[16] The fact that children who had seizures during childhood often ceased to have attacks after puberty, led to the conclusion that seizures disappeared after intercourse. Thus, early authors thought to hasten the cure by "doing malice to the nature of children by unseasonable coition."[15, 16]

Continence with retained semen was thought to cause epilepsy.[6] Retained menstrual blood was thought to be another cause.[1] Perhaps the worst idea of the cause of epilepsy was held by those who attributed it to masturbation.[17] This belief continued to the time of Gowers[10] and is held by some even to the present.[18]

Aside from the general views of the hereditary nature of epilepsy, the Talmud states that epileptic children will result if "parents have intercourse before a burning candle, in a room near which many women work at a hand mill, immediately following defecation, after the letting of blood or recumbente marito. Also if they have intercourse with a child at the foot of their bed."[6, 8] Intercourse during mensus,[2] onanism,[19] difficult menstruation or amenorrhea,[7] sometimes even pregnancy[1]—all these have been stated as causing epilepsy. It is interesting to note that from the belief that seizures originate in the uterus we get our present day word "hysteria."[1]

Epilepsy has been considered a visitation of the gods, the loss of the soul, and the possession by a devil.[1] There were many things that could be done to bring these states to an end and more to keep them from happening. There were so many different evil spirits indicted that a cure for one devil often would be useful for a number of different evil spirits. It is rather odd that while believing epilepsy to be caused by a demon, an occasional epileptic, during the same period in history, was considered to have been visited by a god (a rare privilege), which inclined him to prophetic activities and moving experiences. Some people believed that this caused the greatness of certain famous epileptics, such as Matthew, Napoleon, and Dostoievski.[20, 21]

The simplest way to rid an epileptic of a demon was to bore a hole through which the demon could escape. Trephined skulls dating from the neolithic period have been found and are said to have been trephined for purposes of releasing demons.[22] Trephining as a treatment for epilepsy is recorded down through the ages.[9, 23, 24, 25, 26]

Human skull bones have long been reputed to do a fine job of curing epilepsy. There were many different manners of preparing these bones; charred, powdered, and in various mixtures.[2, 6, 27] The bones that were supposed, by Paracelsus, to be the most useful were small suture bones of the skull,[28] the idea, apparently, being an oral prefabricated demon escape hatch.

Many ways were used to pin point the particular demon or god causing the trouble: "If a patient imitated a goat or ground his teeth, or if the right side was convulsed, Hera, the mother of the gods, was the cause. If the patient spoke in a sharper or more intense tone then he was likened to a horse and Poseidon was the cause. If any excrement was passed, the appellation of Euodius was at fault; if it was passed in small dense masses, like birds, Apollo Noimus was the cause. If foam was emitted by the mouth and the patient kicked, then Ares got the blame. Night tremors, frightful apparitions, and running away were plots of Hecate and Heroes."[6]

Hippocrates even goes further and cautions against wearing black as that is the color of Hecate.[7]

Aretaeus says that epilepsy is called the sacred dis-

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1. Recommended Dietary Allowances, Washington, D. C., National Academy of Sciences—National Research Council, Publication 302, 1953.
2. Co Tui: Review: The Fundamentals of Clinical Proteinology, J. Clin. Nutrition *1*:232 (Mar.-Apr.) 1953.
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ease not because it was caused by gods but because only gods could heal it,[15] and Bernard of Gordon says of his inability to cure epilepsy, "I am ignorant, but God has knowledge."[2]

Before more demon causes of epilepsy are enumerated, it might be well to cite a method used to ward off epilepsy. Pliny says, "In cases of epilepsy we spit, that is, we throw back contagion."[16] The idea of spitting is so strong that epilepsy was even called the spitting disease, and references are found even to the present day of the good of spitting.[18]

In Ethiopia care was taken lest the shadow of an enemy fall across the patient, causing epilepsy. Mohammedans believed epilepsy was caused by jinns, while the Turks believed the attacks were from spirits who were in love with the same one as the patient and the spirits were trying to throttle the patient in revenge.[29]

Russian peasants believed a patient in a seizure was overpowered by a demon, and they beat the patient unmercifully with a sacred chain in order to force the demon to leave.[6] The Hindus believed that Kurkura, the dog-demon, was the source and gave a nice prescription for a cure. This cure consisted of incantations, beating of gongs, and a ritual, all the while pouring a mixture of curds and salt water over the patient.[29]

From the Christian world there are about as many fine methods of driving out the devils. From the Bible (Matthew XVII, 18) there is recorded the story of a boy who was brought to Jesus for help, "And Jesus rebuked the devil and he departed out of him and was cured from that very hour." This passage from the Bible has evoked a great deal of discussion through the ages. At one time it was quoted to show that epilepsy could be caused by demons.[1]

From Scot we have this cure: "Take the sicke man by the hand, and whisper these wordes softlie in his eare, I conjure thee by the sunne and moone and by the gospell of this daie delivered by God to Hubert, Giles, Cornelius, and John, that thou rise and fall no more."[30]

Another suggestion was that if you chance upon a patient in a seizure, bend down and whisper in his ear three times, "Gaspar bears the myrrh, Melchior the frankincense, Balthasar the gold. Whosoever bears with him the names of these three kings is delivered from epilepsy by the holiness of Christ."[2] Besides curing the seizure this verbal formula was said to cure the disease if the patient would fast, pray, and write the above on a paper and wear it around his neck. This probably was thought to cure the disease as the three kings *fell down* before Jesus.[2]

The first man to see a person in a seizure was to take off his right shoe, urinate in it, swirl it around in a mixing motion, and give it to the patient to drink. John of Gaddesden goes even further and reports that the reason the first person seeing a seizure is the one to do the ritual is because of purely psychological reasons,[26] a bizarre finding in that age.

There were many patron saints to whom epileptics appealed. There was a ritual of attending Mass three times, saying certain prayers, and at times visiting the saint or his grave. Among these saints were St. Valentine, St. Nicetius, and St. Bibians. Some saints even passed out herbs, too.[2, 6, 26, 27]

Another method of removing demons is quoted by Talbot. It shows that people, even in one method of treatment, did not put all their trust in the saint to get rid of the epilepsy.

"The patient first washes his limbs in a sacred well near the church, drops fourpence into it as an offering and walks three times around it, and repeats the Lord's prayer three times. Then a fowl, which was a cock or a hen, according as the patient was a man or woman, was put into a basket and carried first around the well and afterwards the church. Next the sufferer entered the church and lay down under the communion table till break of day. After that he offered sixpence and departed, leaving the fowl in the church. If the bird died, the sickness was supposed to have been transferred to it from the man or woman who was now rid of the disorder."[9]

The concept that epilepsy was transferable and contagious is seen throughout history, even to the present day.[13, 18]

During the middle ages epileptics were not permitted to sell articles of food or drink.[6] While epileptics were often looked upon as mentally ill, it was during this time that they were isolated from mental patients. This was done, not for the benefit of the epileptics, but for the mental patients, "since the sight of one epileptic attack may suffice to make a healthy person epileptic, how much greater is the danger for the mentally deranged who are so much more impressionable."[31]

Willis of the 17th century, who is reputed to be one of the pioneers in the evolution of modern neurology, is quoted as saying of epilepsy: "As often as the devil is permitted to affect miserable mortals with his delusions, he is not able to draw more evil arrows from any other quiver, or to show miracles by any better witch, than by the assaults of this monstrous disease."[23]

The moon goddess, Phoebe, was early in history thought to cure epilepsy,[15] but later she was left out and the moon itself was accused. As the moon waxed and waned, so also did epileptics have attacks. As late as 1861 Sieneking wrote, "Lunar influences are still upheld by some physicians of repute, and the

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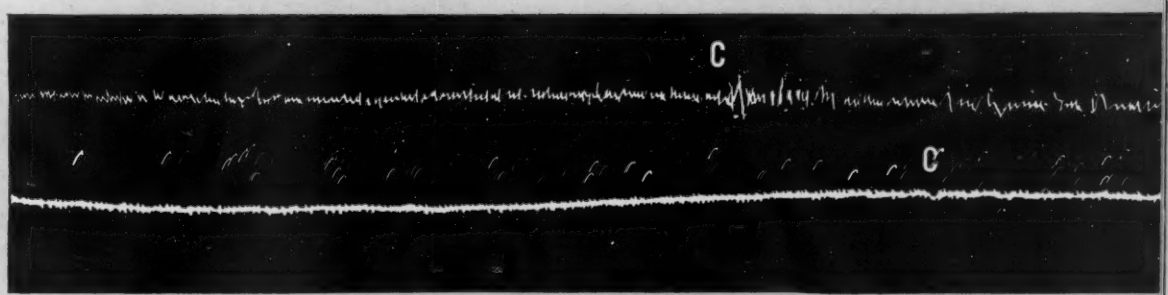

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1. Dickel, H.A., et al.: West. J. Surg., April, 1956.

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belief is generally in vogue among the public. Epileptics were termed moony or moon struck."[32] Pliny says that the moon attracts water to the brain, causing epileptic attacks.[16]

Richard Mead in 1704 wrote what was considered a learned and valuable treatise on the influence of the moon on humans. His work was useful as he allegedly could tell when sailors of the Royal Navy were going to have seizures "with tolerable certainty."[33] The belief that epilepsy was worse during the moon's phases was scientifically discredited in 1854 by Moreau's paper, for which he received a prize from the Paris Academy of Medicine. He showed of the 42,637 attacks observed by him, by actual count 26,313 were between the phases of the moon and only 16,324 during its phases.[33] And that takes care of the moon.

Realizing that these are but a few of the views about demons, gods, and epilepsy, we pass to other superstitions.

The highest order of animals used in the treatment of epilepsy is man. Human bones have been mentioned in connection with demons. Human blood has been used often in the treatment of epilepsy. Pliny tells that in ancient Rome epileptics drank the blood of gladiators "—as though from living cups, which looks very gruesome if there were also wild beasts in the arena. They think it most valuable to sip the blood still warm, still flowing from the wounded themselves and thus to imbibe the breath of life immediately from the fresh opening."[16]

Aretaeus Cappadonious describes epileptics assembled at the place of execution to lap up the blood of a just beheaded criminal.[15] During the middle ages the act was followed by whipping the patients and making them run away as fast as they could after drinking the blood.[6]

Pliny advises rubbing the feet of an epileptic with virgin's menstrual blood as a means of reviving a person from a seizure.[16]

In Brunswick an epileptic child was to drink some of his own father's blood and in Denmark some of both his father's and his mother's blood.[6] The executioners in Poland are said to have had an extra income by selling the blood of beheaded criminals as late as 1909.[6]

The brain of a child or human bone marrow was also advised by Pliny,[6] and Kanner lists the following uses of human parts in the treatment of epilepsy: In the 13th century a dead man's tooth was believed helpful. The afterbirth of a first born child and scrapings of the vertebrae of a man killed by violence were prescribed. In Germany human perspiration is believed to bring a person out of seizures. From Maryland a strange magic procedure in which the patient's hair serves as a means to transfer the disease from him to an inanimate object: Bore an augur hole into a piece of wood on a level exactly with the top of the sufferer's head; stuff into the hole some of his hair and plug it up.[6]

Enough of homosapiens and on to other forms of life used in past treatments of epilepsy. If the whole of the animals used for treatment were assembled, one would have quite a zoo. Particular reference was made to the whole animal as the following are some parts of animals that were prescribed: brain, heart, milk, liver, gall, testicles, urine, dung, claws, teeth, hide, horns and feathers.[1, 6]

Kanner gives an alphabetized list of animals, about which he says, "I wish to emphasize that it is only a selection of the vast list of animals employed in the folk cure of epilepsy which is at my disposal."[6] The list includes 67 different animals.

Now a few prescriptions in which animal parts were used will be listed.

Pliny advised the following prescriptions: Eat the heart of a black mule on the first or second day of the new moon; or ass's testicles drunk in ass's milk; eat a young dog still suckling, with wine and myrrh, after the head and feet have been removed; hawk's liver, hen liver, cock's testicles, pig's testicles in pig's milk. Strong persons may take 21 houseflies in a liquid medium; weak persons should not take so many. Lamb's gall, vulture liver drunk with vulture blood three times a day for seven days, or wear the heart of a vulture, caught while feasting on a human corpse, around the neck.[16]

St. Hildegard of Bingen gives the following: Take four parts of dried mole's blood; two parts of powdered duck's bill; one portion of the powdered claws of a goose, minus the skin and flesh. These constituents are bound together in a cloth, placed for three days near a mole track, for such earth is more wholesome—then they are put near ice to cool and then in the sun to dry. Cakes are then to be made with this powder and liver of some edible animal or bird and a little flour and cummin seed, and eaten for five days.

St. Hildegard even explains why these ingredients are used. She says: "Mole's blood is used because the mole sometimes shows himself and sometimes hides, like epilepsy itself. Duck's bill is added because it touches both pure and impure things with its bill, it is repugnant to this disease which is sudden and silent. Claws of a goose are advised for much the same reason and the claw of a goose rather than a gander because the female is the more silent of the two."[19]

Antonius Guainerius has this prescription to offer: "When a fit begins, kill a dog, remove its gall and give it to the patient in whatever way you can." Or: "For epilepsy, take a frog's gall and wrap it in a cab-

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1. Spies, T. D., et al.: GP 12:73, No. 1, 1955. 2. Boland, E. W.: J.A.M.A. 160:613, 1956. 3. Gillhespy, R. O. Lancet 2:1393, 1955.

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bage leaf, dry it in the sun to make a powder and give to the patient mixed with a good wine."[27]

Bernard of Gordon recommended that the cuckoo, after it has been placed in a jar and dried in an oven, be ground and given to a patient. This was said to be good because the cuckoo itself has fits and thus the poison will counteract the disease.[17]

The following quotation from Kanner is to show that wood lice were still being used in the 20th century: "In 1925 the old wife of a German innkeeper was accused of selling to epileptics white wine with wood-lice, that must have died in the wine; she was acquitted because the court assumed she believed in the efficacy of the remedy and therefore acted bona fide. . . ."[6]

Of the things vegetable that have been used, the one that has been recommended the most and for the longest period of time is mistletoe. The Hippocratic papers mention it, as does every other author writing of epilepsy. Even Gowers himself in 1881 had this to say about mistletoe: "This old remedy for epilepsy, praised by Paracelsus and by a considerable number of writers since his time, especially Calbatch and Frazer, I have tried in a considerable number of cases, but with beneficial results only in a case in which the attacks were apparently hysteroid."[10]

J. G. Frazer, in his *Golden Bough,* gives this explanation of the virtues of mistletoe: "As mistletoe cannot fall to the ground because it is rooted on the branch of a tree high above the earth, it seems to follow as a necessary consequence that an epileptic patient cannot possibly fall down in a fit as long as he carries a piece of mistletoe in his pocket or a decoction of mistletoe in his stomach. If I had not tried it, as far as the limited opportunities of an individual would admit, if I had not found it efficacious in epilepsy, even beyond my most sanquine expectations, I would not presume to offer it as worthy of the most serious attention."[36]

Pliny states that mistletoe works much better if gathered on the first day of the moon without the use of iron and if, when collected, it was not allowed to touch the ground.[16]

Peony root or seed, worn around the neck or taken internally, is recommended by Galen. Garlic also has a long history in the treatment of epilepsy, dating from the fifth century and mentioned in the Bower manuscript.[6]

Blockwick prescribes elder as a treatment for epilepsy. He says: "If in the month of October, a little before the full moon, you pluck a twig of elder, and cut the cane between two of its knees or knots, in nine pieces and these being bound in a piece of linen, be it in a thread so hung about the neck that they touch the spoon of the heart or the sword formed cartilage; and, that they stay more firmly in place, they are bound thereon with a linen or silken roller wrapt around the body, till the thread break of itself. The thread being broken and roller removed, the amulet is not at all to be touched with bare hands, but it ought to be taken hold of with some instrument, and buried in a place that no body may touch it."[37]

In Russia lily of the valley is used; in China the rhammus root is preferred, and in Sumatra betel is given for epilepsy.[6]

P. Borel gave powdered soap-wart seed for three months at the time of the new moon, because the plant has a signature of its property, "for if rubbed in water it emits a soap like foam and is therefore profitable to the frothing epileptics."[38] Bernard of Gordon gives the following as having been used: endive, chicory, lettuce, hyssop, mountain laurel, physalis, anise, maratium, diagrydum, bdeliu, rue, cloves, absenthe, and pyrethium.[2]

It would seem that if the plant is known, there is somewhere a prescription for it or a warning against the use of it.

Of the things mineral St. Hildegard says about the gem agate, "It should be soaked in water for three days at the full moon; the water should be slightly warmed and then preserved and all the patient's food cooked in it until the waxing of the moon; the gem should be placed in everything he drinks." This procedure was to be followed for ten months.[19]

Jade was worn by the Greeks, and emeralds were recommended by the Arabs, both prophylactically and therapeutically.[6] Paracelsus recommends the emerald also.[28] Some believed if the violence of the disease were such that it could not be overcome by the gem, the gem broke into pieces.[39] Seligmann states that South American Indians of the Orinoco River place a piece of green fledspar in a wound which has been incised for the purpose in the skin of an epileptic; after the scar heals, the patient will be cured.[40] Paracelsus recommends white coral to be worn as a cure.[28]

The swallow stone, which is found in a swallow's stomach at the beginning of autumn in the first quarter of the moon, reduced to powder and given in a liquid, was said to cure epilepsy.[2]

Antonius Guainerius practically guarantees a cure from the following prescription: Place some umbilical cord from a new born baby in a gold ring that has an emerald on it. As long as it is worn epilepsy cannot strike.[27]

Of various metals that were used, iron filings were said to be good taken internally, or an iron nail driven at the spot an epileptic first fell would rid him of the evil.[16]

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A dramatic case in point is mastoiditis in children. Surgery was often able to save the child's life, but sometimes left him with a deaf ear. Nowadays, the doctor is often able to cure him with perhaps $25 worth of potent new medicines, and thus save the expense of an operation and hospital stay.
But most important, these new drugs are saving children's lives—by the millions. Cases, the four most deadly child-killers were diphtheria, whooping cough, scarlet fever, and pneumonia. Nowadays, the number of deaths from all of these causes combined is actually less than the number of children killed by accidents.
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cow's urine, and lastly in the juice of okra. Melt the purified metal in an earthen crucible, adding to it powder of tamarind and banyan tree barks in the proportion four to one. Stir and rub then with an iron ladle. Mix with the powder an equal quantity of talaka (terasulphide of arsenic) and triturate in an acid juice. Expose it to fire; again add one-tenth of its quantity to talaka, and again rub it and put it over a fire. Repeat the process ten times until the metal is reduced to bhasma.[41]

Gower also wrote, "Zinc unquestionably deserves some of the repute which it has enjoyed for more than a hundred years as a remedy for epilepsy."[10]

Table salt, antimony, and asphalt have also been used.[6]

Interesting points in regard to the use of minerals are the cramp rings and touch coins or sacramental shillings. Cramp rings were especially made rings which were to protect the wearer from epilepsy. The custom is said to have started when Edward the Confessor brought back from Jerusalem a ring thought to cure the epilepsy of those touching it. Later other rings were made; the best were supposedly those made from a nail or a screw from an old coffin, and certain prayers were said over them. The king then rubbed the rings between the palms of his hands, saying, "Sanctify, O Lord, these rings and graciously bedew them with the dew of thine benediction and consecration, and hallow them by the rubbing of our hands which thou hast been pleased according to our ministry, to the end that what the nature of the metal is not able to perform may be wrought by the greatness of Thy grace."

The rings were then passed out to the patient to be worn.[42]

Of the sacramental shillings which were either carried or worn, Kanner quotes the *London Times* of March 7, 1854: "A young woman, living in North Devon, having for some time past been subject to periodical fits of illness, endeavored to effect a cure by attendance at the afternoon service at the parish church, accompanied by 30 young men, her neighbors. Service over, she sat near the porch of the church and each of the young men as they passed out dropped a penny in her lap, but the last instead of a penny gave her half-a-crown, taking from her the 29 pennies. With this half crown in her hand, she walked three times around the communion table, and afterwards had it made into a ring by the wearing of which she believed she would recover her health."[6]

Silver nitrate was used in the treatment of epilepsy, sometimes even to the point of argyria. Silver was used because silver is the color of the moon, and the moon was said to cause epilepsy.[8]

Other forms of treatment included bleeding the patient through various portions of his anatomy, different places depending upon the nature of the seizures.[26] The use of cautery at different levels of the body has a long history.[8, 28] Counter irritants were used, including cantharides even to the point of blistering the parts.[8]

One last prescription: "Take a drachm of camphor, divide it into nine parts and give it to the patient to drink in his own urine, then bind on his navel a piece of toasted rye bread and let him sweat in a warm room in a bed well covered over."[43] No explanation is given with the above prescription.

In the words of Antonius Guainerius on the treatments of epilepsy: "If you do these things, it would be a very malignant epilepsy which would not forsake the patient after observing the prescriptions advised."[24]

In conclusion let it be said that, bizarre as these "treatments" of epilepsy are, the people of their times took them in good faith and apparently got some relief. As has been said, the psychotherapy of getting a prescription tends to keep an epileptic from having as many seizures for a while.

It would seem wise indeed to keep this in mind and not to cast too many aspersions on these superstitions of treatment. One day in the future the very drugs being evaluated today, such as hibicon, phenuron, and mysoline, may appear in a paper on superstitions in the treatment of epilepsy.

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BOOK REVIEWS

Peptic Ulcer: Diagnosis and Treatment. By Clifford J. Barborka, M.D., and E. Clinton Texter, Jr., M.D. Published by Little, Brown and Company, Boston. 290 pages. Price $7.00.

This monograph is complete, concise, and straightforward. It is excellent reading for those with a general interest in medicine and those with a special interest in gastroenterology. The discussion of pathophysiology is comprehensive and accurate. Principles of medical management are outlined in the light of the underlying known factors important in the clinical picture of peptic ulcer.

Prevention of recurrence and complications is emphasized, an aspect which is often slighted in the over-all management of the ulcer patient.

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This book might well be placed on the medical students' reading list along with his textbook of medicine.—*A.P.K.*

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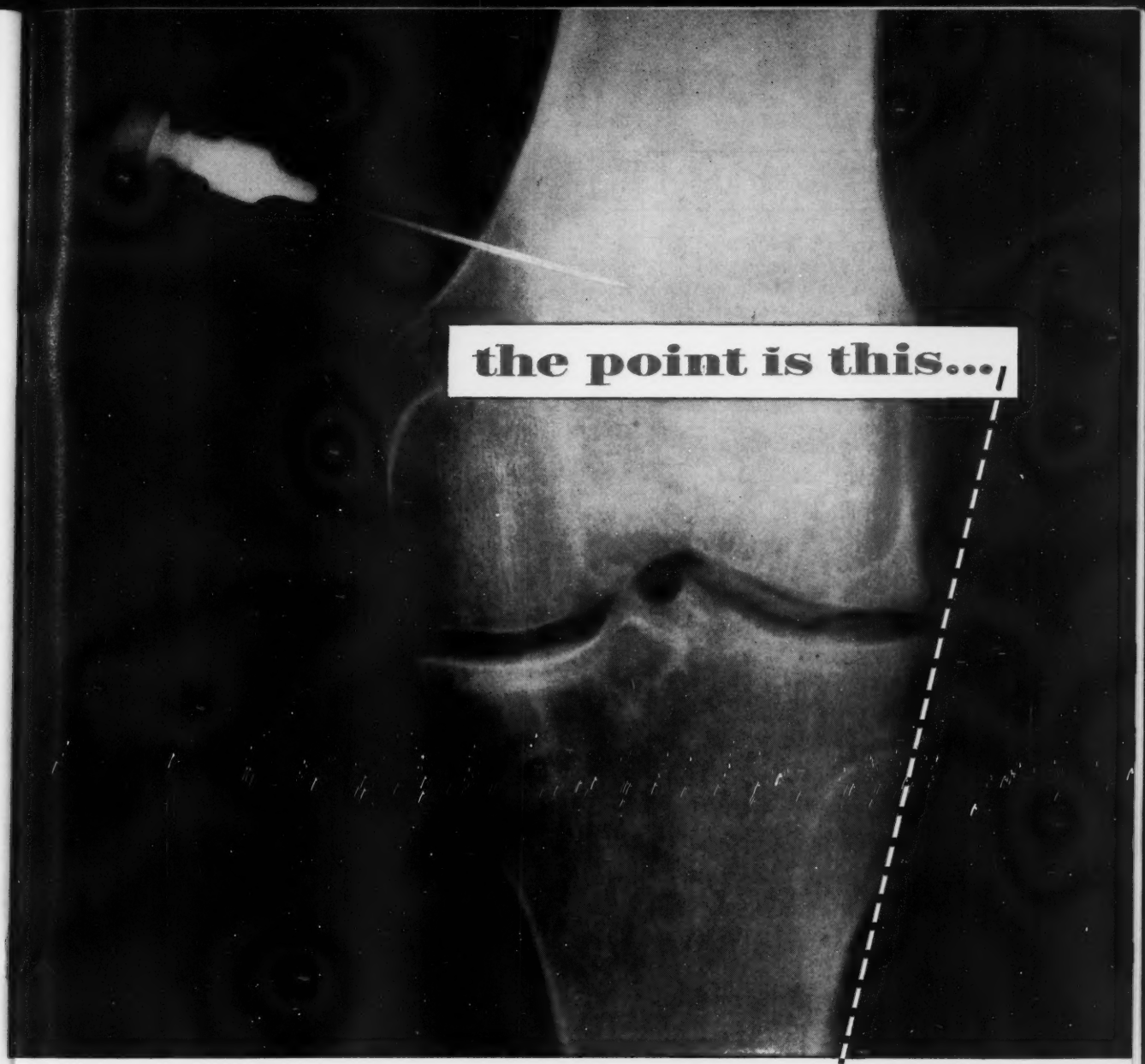

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COUNTY SOCIETIES

"Hospital Emergencies" was the subject of a panel discussion which formed the program for a meeting of the Shawnee County Society on March 5. Dr. Howard E. Roberts was moderator, and the participants were: Dr. Robert P. Woods, Dr. Robert M. Brooker, Dr. James A. McClure, Dr. G. Bernard Joyce, and Dr. Leslie L. Saylor.

Dr. Conrad M. Barnes, Seneca, president of the Kansas Medical Society, was speaker at a meeting of the Shawnee County Society held on April 2.

Members of the Geary County Society were hosts at a meeting of the Golden Belt Medical Society held in Junction City on April 5. A clinicopathological conference, presented by Dr. Vernon E. Wilson of the University of Kansas Medical Center, formed the program, followed by a dinner.

Members of the Pratt County Society were entertained by their wives at a buffet supper at the Park Hills Country Club on March 30 in observance of Doctor's Day.

Vice Admiral Ross T. McIntire (retired), executive secretary of the International College of Surgeons, was speaker at a meeting of the Sedgwick County Society held in Wichita on March 6. His subject was "Blood and Its Derivatives."

The group held its next meeting on April 3. At that time the speakers were Dr. Ralph C. Moore of Omaha and Dr. Charles Marsh of Valley, Nebraska. The subject of the program was "Medical Aspects of Highway Accidents." Dr. Conrad M. Barnes, Seneca, also spoke to the group.

"Income Tax Problems Relative to the Medical Profession" was the subject of a talk given to the Wyandotte County Medical Society at a meeting held on March 20. The speaker was Mr. George Sinderson, C.P.A.

The Southeast Kansas Medical Society, composed of physicians in nine counties, met at the Armory in Iola on March 15 with members of the Allen County Society as hosts. Dr. Clarence H. Benage, Pittsburg, was principal speaker and others on the program included Dr. Conrad M. Barnes, Seneca, president of the Kansas Medical Society, and Mr. Oliver E. Ebel, Topeka, executive secretary.

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GEORGE V. BRINDLEY, M.D., Temple, Texas, President, American Cancer Society

CHARLES L. DUNHAM, M.D., Washington, D. C., Director, Division of Biology and Medicine, Atomic Energy Commission

CHARLES HUGGINS, M.D., Chicago, Ben May Laboratory for Cancer Research

DWIGHT H. MURRAY, M.D., Napa, California, 1956-57 President, American Medical Association

EUGENE P. PENDERGRASS, M.D., Philadelphia, Professor of Radiology, University of Pennsylvania School of Medicine

GRANT H. SANGER, M.D., New York, Francis Delafield Hospital, Cancer Research Laboratories

JOHN R. SCHENKEN, M.D., Omaha, Nebraska Methodist Hospital

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A meeting of the Lyon County Society was held at Emporia on April 2. Dr. Robert G. Rate, Halstead, discussed "Cancer of the Lung."

Physicians of the Russell County Society entertained members of the societies in Ellis and Ellsworth counties at a meeting at Russell on April 5. Two speakers from Kansas City, Missouri, provided the program. Dr. Alexander Moruzi spoke on surgery of the stomach, and Dr. Milton H. Noltensmeyer discussed different types of anesthesia.

Cost of Hospital Care

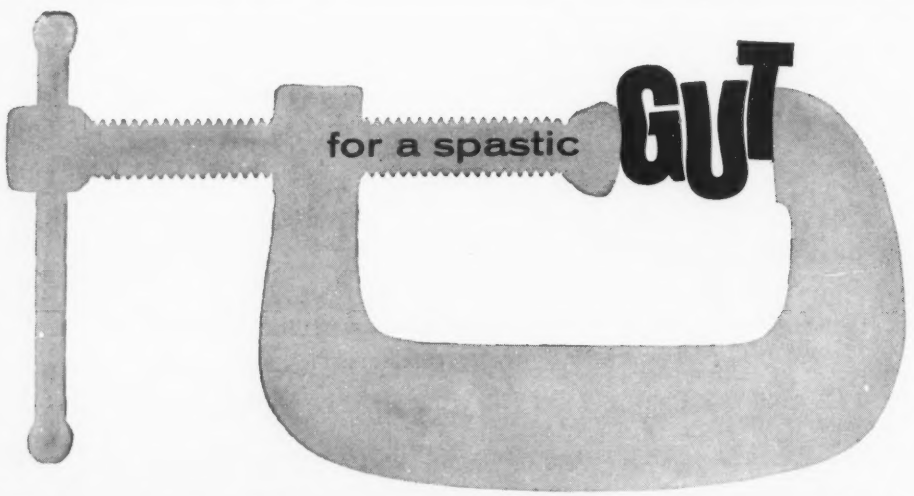
A prediction that hospital costs would "continue to increase at about five per cent annually for many years" was made recently by Mr. Ray E. Brown, president of the American Hospital Association. The prediction is based on his belief that there will probably be no significant decrease in the general economic situation.

"Only by the best efforts of hospital boards, administrators, medical staffs, and all members of the hospital team can costs be held within that level of increase," he said. "Whatever answers there are to the problem of hospital costs must be found in the area of personnel budgets.

"The nature of the hospital's work provides little opportunity for productivity gains. Nonetheless, hospital salary levels are affected by general salary levels. Under such circumstances every round of salary increases constitutes a direct increase in hospital costs."

Increases in hospital services calling for added equipment and personnel are another factor in the rising cost picture. The largest increase in personnel is attributable to new services. Cursory studies made on this question indicate that the number of routine procedures per patient day has increased more than 30 per cent in the past nine years.

The cold war against tuberculosis calls for a clear-cut program for the future. It is regrettable, that in our satisfaction with the fall in death rates, we may have given the impression that tuberculosis is conquered. In fact, some in authority have said that the fight is as good as over and that there will be no tuberculosis problem in ten to twenty years. This breezy optimism is founded on lack of knowledge and misunderstanding of the problems involved. Tuberculosis, while it has lost many of its death-dealing features, is still the greatest single cause of loss of man-hours in young people and still disrupts thousands of homes.—*George J. Wherrett, M.D., Nat. Tuberc. A. Tr., May, 1954.*



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ANNOUNCEMENTS

Physicians are invited to attend the seventh annual Dr. F. G. Thompson, Sr. lectureship at the Thompson, Brumm and Knepper Clinic, St. Joseph, Missouri, at 8:15 p.m. on Monday, May 21. Dr. William Dameshek, professor of clinical medicine at Tufts College Medical School, Boston, will discuss "The Spleen and Hypersplenism."

The Children's Hospital of Philadelphia, 1740 Bainbridge Street, Philadelphia, will present three courses in May and June: (1) Pediatric Advances, May 28 through June 1, tuition $100; (2) Practical Pediatric Hematology, June 4, 5, and 6, tuition $60; (3) Blood Group Incompatibilities and Erythroblastosis Fetalis, June 7 and 8, tuition $50.

The 22nd annual meeting, American College of Chest Physicians, Hotel Sherman, Chicago, June 6-10. Write the College, 112 East Chestnut Street, Chicago 11, Illinois.

Fifth annual symposium for general practitioners on tuberculosis and other chronic pulmonary disease, Saranac Lake, New York, July 9-13. Twenty-six hours credit by A.A.G.P. Fee $40. Write Dr. Edward N. Packard, P. O. Box 262, Saranac Lake, New York.

July 15 is closing date for Ames Award Contest for papers on gastroenterology written by fellows in gastroenterology, residents, and interns. Winning entries, carrying awards of $400 and $250, to be selected by Research Committee, American College of Gastroenterology. Address inquiries to College, 33 West 60th Street, New York 23, New York.

Applications being accepted now and until October 1 for candidates for certification by American Board of Obstetrics and Gynecology. Address inquiries to Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6, Ohio.

The 84th annual meeting of American Public Health Association and 40 related organizations, Convention Hall, Atlantic City, November 12-16. Write the Association, 1790 Broadway, New York City.

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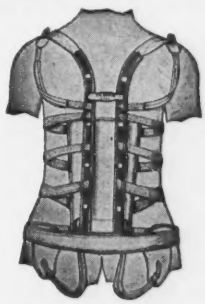

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Hospital Cancer Programs

A properly functioning registry of cancer patients is a requirement for approval of a hospital's cancer program by the American College of Surgeons under new regulations which are now effective, the Committee on Cancer of the College announced recently.

The program considers a hospital's cancer activities only and is entirely separate from the Joint Commission on Accreditation of Hospitals, of which the American College of Surgeons is a member organization.

Minimum requirements for approval of a cancer program in a general hospital conducting organized cancer clinical activities include a cancer registry, cancer consultation, and treatment service. Smaller hospitals are required to maintain only a registry of all cancer patients. All types of programs must be supervised by a committee of the hospital's medical staff.

The registry must include the name and address of every patient upon whom a diagnosis of cancer is or has been previously made, with adequate diagnostic information and an abstract of the clinical record. Annual follow-up notes must be maintained as long as the patient remains alive. College requirements also specify procedures for maintaining the registry and methods of developing the case abstract file. Surveys are made regularly to make certain that standards are maintained. Six hundred twenty-five hospitals in this country are now approved.

Paintings on History of Medicine

Parke, Davis and Company recently announced completion of arrangements for 40 oil paintings depicting major events and personalities in the medical profession during the past 5,000 years. The artist, Robert A. Thom, and the editor, George A. Bender, will require at least five years to complete their work. The new series will complement the "History of Pharmacy in Pictures," commissioned by Parke-Davis in 1951 and soon to be finished.

Appointments to Faculty

The following appointments to the staff at the University of Kansas School of Medicine were announced recently: Dr. Charles M. Poser, Dr. Max Musgrave, Dr. Marjorie Spurrier Sirridge, Dr. Tom Chin, and Dr. Albert Decker as instructors in medicine; Dr. Truman B. Schertz, instructor in ophthalmology.

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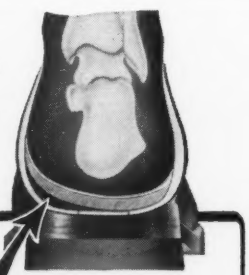

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Health Insurance Coverage

Nearly two out of every three men, women, and children in the United States now are protected by voluntary health insurance, according to the ninth annual survey made by the Health Insurance Council, New York. Figures used in the report were those of December 31, 1954.

Progress in coverage during the past year indicates that at this time some 104 million persons have voluntary protection against hospital expenses, 89 million have surgical expense insurance, and 50 million have insurance for medical expense.

Benefit payments on health insurance claims reported by the survey for 1954 exceeded $2.7 billion, a gain of 11 per cent over the previous year. More than half went to help meet hospitalization expenses of beneficiaries, and more than $730 million paid for surgical and medical care. Benefit payments to policyholders by insurance companies for loss of income due to disability were in excess of half a billion dollars last year.

Of the aggregate benefit payments in 1954 by all forms of voluntary health insurance, 56 per cent came from insurance companies. The dollar amount paid by the companies was more than $1.5 billion, including loss-of-income benefits.

Blue Cross and Blue Shield type plans paid more than $1 billion, or 39 per cent of the total. Various independent plans accounted for the remaining 5 per cent of the total.

On December 31, 1954, a total of 101,493,000 Americans had hospital expense protection. This represents an increase of 4.3 per cent during that year, a rate of increase which is over 2½ times the rate of population growth in the same period. Since the beginning of 1941, the number of persons with hospital expense protection has multiplied nearly 8½ times.

Nearly 86 million persons had surgical expense protection by the end of 1954. This represents an increase of 6.1 per cent over the previous year. Ordinarily, people with surgical coverage also have hospitalization protection. So, up to 85 per cent of those with hospital expense protection also had surgical coverage. Since 1941, the number of persons with surgical insurance has multiplied about 16 times.

Regular medical expense coverage increased by more than four million persons, or nearly 11 per cent during 1954, to give a total of more than 47 million who have this protection against the cost of nonsurgical medical care by their doctors. People with medical expense protection usually have hospital and surgical protection as well.

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*Moyer, J. H., and Hughes, W. M.: J. Chron. Dis. 2:678, 1955.

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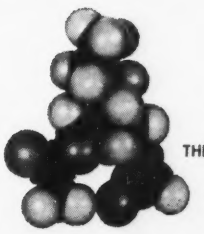

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